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Dott. Thomas Vaccari

CURRICULUM VITAE

INFORMAZIONI PERSONALI

Cognome:	Vaccari
Nome:	Thomas
Data di nascita:	25 Marzo 1973

INDICE

EDUCAZIONE E STUDI

-Dal 1992 al 1997 ha frequentato il Corso di Laurea in Scienze Biologiche presso l'Università degli Studi di Milano, indirizzo Biomolecolare; ha conseguito la laurea il 16 Dicembre 1997 con la votazione finale di 110/110 e lode, discutendo una tesi dal titolo "Identificazione del gene *Hmg0,* codificante per una nuova proteina della famiglia HMG1/2" (Relatore Prof. **Marco E. Bianchi**, Correlatore Dott.ssa Monica Beltrame).

-Da Ottobre 1999 a Luglio 2003 ha svolto il Dottorato di Ricerca in Biologia Molecolare all' European Molecular Biology Laboratory (EMBL) a Heidelberg, Germania (Tutor Dott.ssa **Anne Ephrussi**).

-Il 31 Luglio 2003 ha conseguito il titolo di Dottore di Ricerca, presso l'Universita' di Heidelberg discutendo la tesi di Dottorato dal titolo: "Multiple roles of the kinase Par-1 in *Drosophila* cell polarity".

PERCORSO PROFESSIONALE

-Dal Gennaio a Dicembre 1998 è stato borsista presso il laboratorio del professor **Dario diFrancesco** all' Università degli Studi di Milano dove ha lavorato all'identificazione e caratterizzazione di canali pacemaker cardiaci.

-Dal Gennaio a Settembre 1998 ha assolto i propri obblighi di leva, svolgendo il Servizio civile presso il laboratorio dell Dott. **Martino Introna** all'Istituto di Farmacologia Mario Negri di Milano, dove ha contribuito all'uso terapeutico dell'anticorpo bloccante Rituximab nel trattamento delle leucemie.

-Nel periodo Ottobre 1999-Luglio 2003 ha frequentato, come titolare di una borsa di dottorando, il laboratorio di **Anne Ephrussi** all' EMBL di Heidelberg, Germania.

-Dal Luglio 2003 al Giugno 2004 e' stato Postdoc nel laboratorio di **Anne Ephrussi** all' EMBL di Heidelberg, Germania.

-Da Luglio 2004 a Maggio 2009 con una borsa di post dottorato ha lavorato nel laboratorio di **David Bilder** nel dipartimento di Molecular and Cellular Biology all' Universita' della California a Berkeley, USA.

-Da Luglio 2009 e' **Capo laboratorio del Notch Signaling and Tumor Suppression Lab all'IFOM- Istituto FIRC di Oncologia Molecolare di Milano**.

-Nel 2014, con giudizio favorevole unanime delle commissioni, ha conseguito l'abilitazione scientifica nazionale alla funzione di Professore per i seguenti settori concorsuali: 05E2 bio11 seconda fascia, 05F1 bio13 (seconda fascia e prima fascia), 05B2 bio06 (seconda fascia) e 06A1 med03 (seconda fascia).

ATTIVITA' SCIENTIFICA

1) Le principali linee di ricerca seguite dal Dott. Vaccari vertono sulla comprensione della plasticita' del sistema endo-lisosomale e autofagico del loro impatto sulle risposte cellulari e sulla formazione di organi epiteliali. Un breve riassunto di tali line e' fornito qui di seguito (in Inglese):

Understanding how cells accurately use membrane organelles to support epithelial development and homeostasis is one of the most fundamental problems of biology. My work focuses on determining how regulation of signaling at endosomes controls cell polarization, growth, and differentiation events. The endosome has long been regarded as a mere trafficking station on the endocytic route of membrane proteins degradation in the lysosome. However, recent evidence indicates that the endosome affects not only surface and extracellular signals, but also cell-intrinsic messages, suggesting a novel role as a cellular hub for signal transduction. Deciphering the mechanisms of communication and response orchestrated by endosomes is critical to understand their role in development and disease.

Conserved multi-protein complexes have evolved with compartmentalization to direct communications and responses at the endosome. Three of such complexes are critical players in this context: 1) ESCRT (Endosomal Sorting Required for Transport), which in conjunction with the ubiquitin system can downregulate signals by sorting receptors toward degradation; 2) V-ATPase (Vacuolar-ATPase), which controls maturation of endosomes; and 3) SNARE (Soluble NSF Attachment Protein REceptor), which regulate membrane fusion events required for delivery of cargoes to the endo-lysosomal system from other trafficking pathways, such as, for example, autophagy. In association with mTOR and the transcription factor TfEB, V-ATPase also senses levels of nutrients recovered by lysosomes and adjusts the lysosomal compartment to match cellular needs [1].

Mounting evidence indicates that mis-regulation of ESCRTs, V-ATPase, and SNAREs is associated with tumor development and to a host of congenital and late-onset diseases [1]. However, we still know very little about how the activity of these core endo-lysosome regulators impacts cell behaviors. My long-term goal is to gain a deeper understanding of disease-relevant functions of the endo-lysosomal system using *Drosophila* as a model. *Drosophila* harbors a rich set of epithelial organs, for example the imaginal discs, which contain stem-like cells and give rise to adult structures often functionally equivalent those of mammals. A second distinct advantage of *Drosophila* is that it offers a large set of refined genetic tools not available in other model systems or in human cells. Furthermore, its tissues and organs are highly amenable to cell biological and imaging approaches, and its rearing is inexpensive and easily scalable.

Past work: Investigating the role of endo-lysosomal trafficking in cell polarity and tissue architecture.

My work to date has investigated how cells and tissues become polarized during *Drosophila* development. As graduate student in the laboratory of Anne Ephrussi at the EMBL, I used *Drosophila* genetics to study the kinase Par-1, one of the most important and conserved determinants of cell polarity [2, 3]. Upon completion of my Ph.D., I took my expertise in cell polarity and joined David Bilder's laboratory at UC Berkeley for my postdoctoral research. Here, I tackled the problem of how cell polarity contributes to shape the architecture of an epithelial tissue, by performing a mosaic screen in *Drosophila* imaginal discs. Among several other hits, I identified ESCRT-, V-ATPase-, and SNARE-encoding genes as key players in regulating signaling events that control polarization, growth and terminal differentiation during organ development [4, 5]. Strikingly, mutant tissues for these genes displayed tumor-like features revealing that they behave as tumor suppressors. I demonstrated that mis-regulated Notch, a major receptor controlling multiple aspects of cell fate determination signaling, was the underlying cause of uncontrolled proliferation in these tissues [6].

Ongoing and planned research: Understanding how trafficking regulators control Notch signaling and tissue development.

As an independent investigator at IFOM in Milan, I uncovered how ESCRT and V-ATPase regulate Notch signaling in endosomes and how they could prevent tumorigenesis [7-9]. The importance of SNAREs in organ development is unclear. We found a specific function of the SNARE protein Snap29 in controlling the fusion of autophagosomes with lysosomes [15]. This work was among the first to characterize molecularly the point of convergence between endocytic and autophagic trafficking. Intriguingly, *Drosophila* imaginal discs mutants for Snap29 display altered epithelial architecture that is not explained by impairment of autophagy [10]. Considering this, Snap29 might control additional pathways important for correct tissue development. Interestingly, Snap29 is mutated in CEDNIK (CErebral Dysgenesis, Neuropathy, Ichthyosis, and palmoplantar Keratoderma), a rare congenital disease characterized by multi-organ defect and poor life expectancy [11]. Thus, in addition to tumor development, our findings will likely to shed light on CEDNIK pathogenesis.

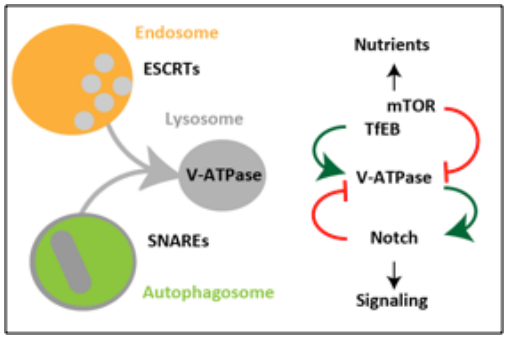

Fig. 1. Schematics of endosomal trafficking and signaling. ESCRT and SNAREs ensure convergence of endocytosis and autophagy (left). V-ATPase controls nutrient and cell fate signaling as indicated (right).

To gain a mechanistic understanding of how the regulatory networks that we identified (schematized in **Fig. 1**) impart cell behaviors important for tissue development and homeostasis, my lab is currently undertaking two major research directions:

1. Investigation of the endocytic control of Notch signaling in health and disease.

Studies in mammalian cells have shown that the mTor/TfEB/V-ATPase pathway acts as a homeostatic loop that uses endo-lysosomal trafficking and autophagy to buffer cells against nutritional stress [12, 13]. Whether or not the mTor/TfEB/V-ATPase pathway influences Notch signaling was not known. We have recently characterized *Drosophila* Tfeb and found that expansion of the endo-lysosomal compartment by TfEB and V-ATPase supports activation of Notch signaling during the establishment of sensory organs precursors (SOPs), a *Drosophila* stem cell model [14]. However, it is unclear if the mTor/TfEB/V-ATPase pathway supports Notch signaling to ensure stereotypic development upon variable cell-intrinsic nutrient levels. To test this, we will raise animals in which the mTor/TfEB/V-ATPase pathway has been genetically manipulated, in nutrient-rich food or upon starvation, and we will measure Notch signaling, using imaging-based quantitative *in situ* reporter assays. We will also follow trafficking of Notch *in vivo*, using recently developed fluorescent lifetime sensors [15], and test for presence of known Notch phenotypes. If a Notch signaling deficit is found upon starvation when the mTor/TfEB/V-ATPase pathway is altered then we will conclude that Notch signaling uses endosomal signaling to buffer nutritional fluctuations, and we will also know which nutrients are sensed.

Another possibility is that the mTor/TfEB/V-ATPase pathway might function to support cell fate determination during development, independently of nutrients. To investigate this, we will analyze changes in the make-up and functionality of the endo-lysosomal system in a number of developmental contexts, such as those in which Notch signaling drives cell fate decisions relevant to cancer. Specifically, we will assess whether endocytic trafficking, lysosomal biogenesis and autophagy are increased during the rapid Notch-dependent proliferation of wing disc cells occurring at early stages of larval development, using *in vivo* uptake assays, available markers or fluorescent sensors, and electron microscopy. These experiments will elucidate how the mTor/TfEB/V-ATPase pathway acts during tissue development. Our studies will be complemented by our established assays in human tissue culture cells to assess the functional conservation of our findings [8]. In addition, we carried out an high content screen in human breast cells in culture to identify genes that control localization of the NOTCH1 receptor, which is extensively mutated in breast and other cancers (**Fig. 2**).

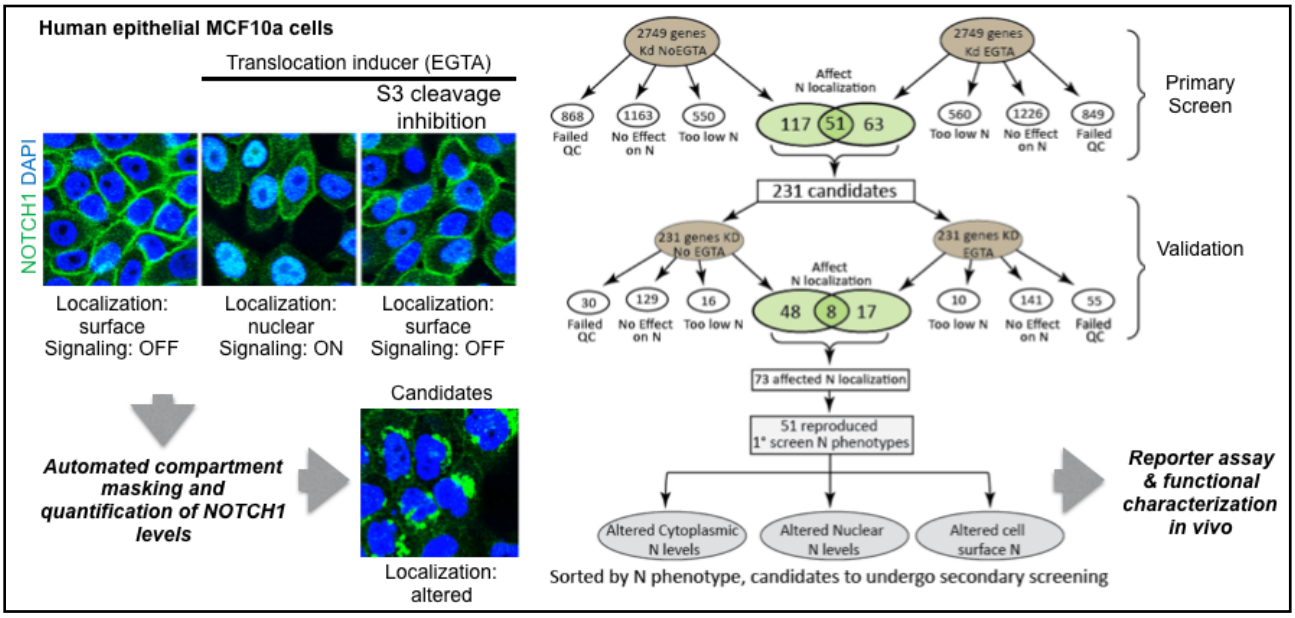

Fig. 2. Endogenous localization assay and screen logic (left). Screen execution and ongoing follow-ups (right).

Surprisingly, we have recently found that V-ATPase expression is elevated in glioblastoma patients [16], suggesting that the mTor/TfEB/V-ATPase pathway might be involved in gliomagenesis. To test this hypothesis, we will use an established genetic model of glioma in *Drosophila*. The model is based on over-activation of the RAS and PI3K pathways in the glial compartment of the larval brain. Such manipulation gives rise to massive over-proliferation and larval death [17]. To determine the involvement of the mTor/TfEB/V-ATPase pathway in gliomagenesis, we will modulate expression of mTor, TfEB and V-ATPase genes and assess the effect on over-proliferation. These experiments will determine how V-ATPase, mTor and TfEB contribute to gliomagenesis.

2. Mechanistic dissection of the role of membrane proteins during cell division.

As part of our effort to elucidate how the SNARE Snap29 controls tissue development, we have made the unexpected observation that Snap29 localizes to kinetochores. Kinetochores mediate the attachment of chromosomes to spindle microtubules at the onset of mitosis. Attachment defects can cause chromosome mis-segregation, which is associated with tumor development. Although many of the protein components participating in kinetochore assembly and function have been identified, it is unclear whether membrane organelles or trafficking factors play a role in this process. We have uncovered that kinetochore assembly is perturbed when Snap29 is depleted or when Snap29-mediated membrane fusion is impaired. Cells depleted of Snap29 form unstable connections with spindle microtubules leading to spindle assembly activation and chromosome mis-segregation and nuclear fragmentation (**Fig. 3**). Snap29 is the first SNARE that localizes at the kinetochore and certain components of the outer kinetochore show similarity with tethering factors involved in trafficking. Thus, our working model is now that kinetochore formation and microtubule attachment to mitotic chromosomes might mimic aspects of trafficking, or that a previously unrecognized contribution of unknown intracellular compartments might exist [18]. To test this model, we will initially identify novel Snap29 interactors that might function at kinetochores via large-scale purification of Snap29 complexes followed by mass spectrometry. The identified factors will be knocked-down as well as localized in Drosophila and human cultured cells. Candidates that are kinetochore-associated or that affect kinetochore formation will be further investigated in Drosophila epithelial tissues, such as imaginal disc, follicular gut epithelia, using live imaging in mutants, or RNAi-lines.

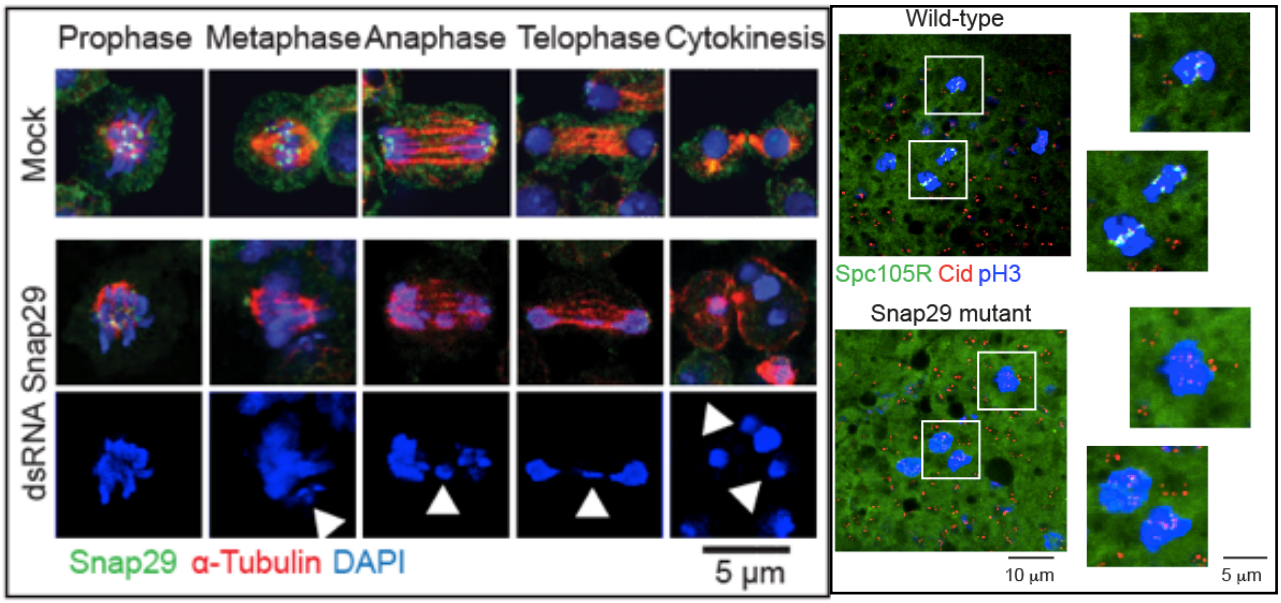

Fig. 3. Snap29 localizes to the kinetochore in *Drosophila*. Snap29 depletion (dsRNA Snap29) results in chromosome congression defects, mis-segregation and formation of cells with micronuclei (arrowheads) (left). Localization of the outer kinetochore component Spc105R (*Drosophila* Knl1) is lost in mitotic cells of Snap29 mutant eye imaginal discs, while the centromeric histone variant Cid (*Drosophila* CenpA) is retained (right).

To investigate the mechanism of Snap29 function at kinetochores, we will express in human depleted or CRISPR cells a Snap29 fused to an Auxin degron domain [19]. We will also express a series of mutants in the domains of Snap29 important for its function in membrane fusion and autophagy. In this way, we will be able to assess the function of Snap29 specifically during cell division. We will also determine which of the Snap29 mutants are defective in kinetochore localization and kinetochore formation. In addition, we will analyze in detail the localization of Snap29 by correlative immuno-electron microscopy. Snap29 mutant forms that are found to perturb kinetochore formation in cells will be expressed in flies and kinetochore formation will be assessed *in vivo*, as explained above Intriguingly, one of the mutants that we have generated affects conserved residues in Snap29 that have been reported to modulate its function in autophagy in response to levels of cellular nutrients [20]. Thus, we will be able to assess whether and how kinetochore formation, and ultimately cell division, is connected with membrane fusion, autophagy, and whether it is under nutrient control. Overall, these approaches will determine how a previously unrecognized membrane dependent pathway controls a crucial step of cell division.

In summary, our future research plans will provide deeper understanding of how trafficking regulators shape epithelial organ form and function, and how their perturbation might lead to diseases, including cancer.

Cited work :

1. Sigismund, S., et al., Endocytosis and signaling: cell logistics shape the eukaryotic cell plan. Physiology Reviews 2012. 92(1): p. 273-366.
2. Vaccari, T. and A. Ephrussi, The fusome and microtubules enrich Par-1 in the oocyte, where it effects polarization in conjunction with Par-3, BicD, Egl, and dynein. Current Biology 2002. 12(17): p. 1524-1528.

3. Vaccari, T., C. Rabouille, and A. Ephrussi, The Drosophila PAR-1 spacer domain is required for lateral membrane association and for polarization of follicular epithelial cells. Current Biology 2005. 15(3): p. 255-261.
4. Vaccari, T. and D. Bilder, The Drosophila tumor suppressor vps25 prevents nonautonomous overproliferation by regulating notch trafficking. Developmental cell, 2005. 9(5): p. 687-698.
5. Menut, L., et al., A mosaic genetic screen for Drosophila neoplastic tumor suppressor genes based on defective pupation. Genetics, 2007. 177(3): p. 1667-1677.
6. Vaccari, T., et al., Endosomal entry regulates Notch receptor activation in Drosophila melanogaster. The Journal of Cell Biology, 2008. 180(4): p. 755-762.
7. Vaccari, T., et al., The vacuolar ATPase is required for physiological as well as pathological activation of the Notch receptor. Development, 2010. 137(11): p. 1825-1832.
8. Kobia, F., et al., Pharmacologic Inhibition Of Vacuolar H+ ATPase Reduces Physiologic And Oncogenic Notch Signaling. Molecular Oncology, 2014. 8(2): p. 207-220.
9. Tognon, E., et al., ESCRT-0 Is Not Required for Ectopic Notch Activation and Tumor Suppression in Drosophila. PLoS ONE, 2014. 9(4): p. e93987.
10. Morelli, E., et al., Multiple functions of the SNARE protein Snap29 in autophagy, endocytic, and exocytic trafficking during epithelial formation in Drosophila. Autophagy, 2014. 10(12): p. 2251-68.
11. Sprecher, E., et al., A mutation in SNAP29, coding for a SNARE protein involved in intracellular trafficking, causes a novel neurocutaneous syndrome characterized by cerebral dysgenesis, neuropathy, ichthyosis, and palmoplantar keratoderma. American Journal of Human Genetics, 2005. 77(2): p. 242-251
12. Settembre, C., et al., TFEB links autophagy to lysosomal biogenesis. Science, 2011. 332(6036): p. 1429-1433.
13. Zoncu, R., et al., mTORC1 senses lysosomal amino acids through an inside-out mechanism that requires the vacuolar H(+)-ATPase. Science, 2011. 334(6056): p. 678-683.
14. Tognon, E., et al., Control of lysosomal biogenesis and Notch-dependent tissue patterning by components of the TFEB/V-ATPase axis in Drosophila melanogaster. Autophagy, 2016. 12(3):499-514.
15. Couturier, L., et al., A fluorescent tagging approach in Drosophila reveals late endosomal trafficking of Notch and Sanpodo. Nature Cell Biology, 2014. 207(3): p. 351-363.
16. Di Cristofori, A., et al., The vacuolar H+ ATPase is a novel therapeutic target for glioblastoma. Oncotarget, 2015. 6(19): p. 17514-31.
17. Read, R.D., et al., A drosophila model for EGFR-Ras and PI3K-dependent human glioma. PLoS Genetics, 2009. 5(2): p. e1000374.
18. Morelli, E., Mastrodonato, V. et al., An essential step of kinetochore formation controlled by the SNARE protein Snap29. EMBO Journal, 2016. 35: p. 2223-2237.
19. Holland, AJ. et al., Inducible, reversible system for the rapid and complete degradation of proteins in mammalian cells. PNAS, 2012. 109(49): E3350-7.
20. Guo, B., et al., O-GlcNAc-modification of SNAP-29 regulates autophagosome maturation. Nature, 2014. 16(12): p. 1215-1226.

2) Contributi Scientifici Piu' Riconoscuti Del Dott. Vaccari E Dei Sui Collaboratori (In Inglese):

1- Endosomal regulation of epithelial development and tumor suppression

We resuscitated the study of Drosophila tumor suppressor genes (TSGs) with the demonstration that ESCRT-mediated endosomal sorting is necessary to prevent tumorigenesis. We explored the functional differences between ESCRT complexes and showed that ESCRT-0 is involved in endosomal sorting but not in tumor suppressive, highlighting a major difference in their activity.

Alfred V, **Vaccari T** (2016) When membranes need an ESCRT: Endosomal sorting and membrane remodeling in health and disease. *Swiss Medical Weekly* 146: w14347.

Tognon E, Wollscheid N, Cortese K, Tacchetti C, **Vaccari T** (2014). ESCRT-0 is not required for ectopic Notch activation and tumor suppression in Drosophila. *PLoS ONE* 9(4): e93987.

Rusten TE, **Vaccari T,** Stenmark H (2012). Shaping Development with ESCRTs. *Nature Cell Biology* 22;14(1): 38-45.

Vaccari T*, Bilder D (2009). At the crossroads of polarity, proliferation and apoptosis: The use of Drosophila to unravel the multifaceted role of endocytosis in tumor suppression. *Molecular Oncology* 3(4): 354-365.

Vaccari T*, Rusten TE, Menut L, Nesis I, Brech A, Stenmark H, Bilder D (2009). Comparative analysis of ESCRT -I, -II, -III function in Drosophila by efficient isolation of ESCRT mutants. *Journal of Cell Science* 122(14): 2413-2423. *co-corresponding

Vaccari T, Bilder D (2005).The Drosophila tumor suppressor vps25 prevents nonautonomous overproliferation by regulating notch trafficking. *Developmental Cell* 9(5): 687-98.

2- Endo-lysosomal control of Notch signaling

We untangled long-standing controversies concerning the regulation of Notch signaling by endocytosis, showing that in fly tissue the activating S3 cleavage of Notch occurs in acidic endosomal compartments and that the vacuolar-H+ ATPase (V-ATPase) is a determinant of Notch activation. We also showed that V-ATPase is developmentally regulated to support Notch signaling during fly development, and that such process involves a master regulation of lysosome biogenesis and autophagy. The results collectively inform therapeutic approaches to Notch- and V-ATPase-dependent tumors.

Tognon E, Kobia F, Busi I, Fumagalli A, De Masi F, **Vaccari T** (2016). Control of lysosomal biogenesis and Notch-dependent tissue patterning by components of the TFEB/V-ATPase axis in Drosophila melanogaster. *Autophagy* 12(3): 499-514.

Kobia F, Duchi S, Deflorian G, **Vaccari T** (2014). Pharmacologic inhibition of vacuolar H+ ATPase reduces physiologic and oncogenic Notch signaling. *Molecular Oncology* pii: S1574-7891(13): 00160-9.

Petzoldt AG, Gleixner EM, Fumagalli A, **Vaccari T*** and Simons M (2013). Activation of the proton pump, V-ATPase, triggers JNK-dependent cell invasion and overgrowth in a Drosophila epithelium. *Disease Models & Mechanisms* 6(3): 689-700. *co-corresponding.

Vaccari T*, Duchi S, Cortese K, Tacchetti C, Bilder D (2010). The vacuolar ATPase is required for physiological as well as pathological activation of the Notch receptor. *Development* 137 (11): 1825-32. *co-corresponding

Vaccari T, Lu H, Kanwar R, Fortini M Bilder D (2008). Endosomal entry regulates Notch receptor activation in Drosophila melanogaster. *Journal of Cell Biology* 180(4): 755-62.

3- New mechanisms of autophagy and cell division

We explored the molecular requirements for completion of autophagy and detailed the function of endosomal factors in the process. We also discovered a new SNARE protein that specifically controls completion of autophagy and that behaves as a Drosophila tumor suppressor. We have found that the tumor suppression activity of Snap29 is likely to be due to control of kinetochore formation, ensuring appropriate spindle formation and completion of cell division in Drosophila and human cells.

Morelli E, Mastrodonato V, Beznoussenko G, Mironov AA, Tognon E, **Vaccari T** (2016). An essential step of kinetochore formation controlled by the SNARE protein Snap29. *EMBO Journal*. 35: 2223-2237.

Morelli E, Ginefra P, Mastrodonato V, Beznoussenko G, Rusten TE, Bilder D, Stenmark H, Mironov AA, **Vaccari T** (2014). Multiple functions of the SNARE protein Snap29 in autophagy, endocytic and exocytic trafficking during epithelial formation in Drosophila. *Autophagy* 10(12): 2251-68.

Rusten TE, **Vaccari T**, Lindmo K, Rodahl LMW, Sem-Jacobsen C, Wendler F, Vincent JP, Brech A, Bilder D, Stenmark H (2007). ESCRTs and Fab1 regulate distinct steps of autophagy. *Current Biology* 17(20): 1817-25.

3) Partecipazione A Progetti Di Ricerca Con Finanziamenti Competitivi

-Il Dott. Thomas Vaccari ha partecipato come principal investigator (PI) of co-PI alla realizzazione dei progetti di ricerca di seguito indicati:

2015-2017 AIRC Investigator Grant	15954	(PI)	400000 Euros
2015-2017 Cariplo Young Investigator Grant		(co-PI; PI: V. Vaira)	250000 Euros
2014-2016 Telethon Investigator Grant	GGP13225	(PI)	250800 Euros
2009-2013 AIRC Start-up grant	6118	(PI)	750000 Euros

4) Organizzazione Di Meeting E Seminari Scientifici

-Il Dott. Vaccari ha co-organizzato o co-organizzera' i seguenti congressi scientifici internazionali:

2018 (in preparazione) Italian Flymeeting
2017 (in preparazione) ABCD Meeting “Organelle Biogenesis and Signal Transduction” Italia (co-organizzatore con L. Lanzetti, E. Van Anken).
(in preparazione) Junior European Drosophila Investigator “JEDI” meeting (co-organizzatore con M. Silies, M. Mueller)
2016 IFOM International Scientific Meeting on chromosome replication and segregation, Milano, Italia. (co-organizzatore con D. Branzei, A. Ciliberto, S. Polo).
2015 DFG Symposium “V-ATPase: A Novel Anti-Tumor Target” Milano, Italia (co-organizzatore con A. Vollmar).
2012 Notch Meeting, Atene, Grecia (co-organizzatore con altri).

-Negli ultimi anni il Dott. Vaccari ha ospitato i seguenti relatori scientifici per seminari SEMM (2 all’anno in media):
N. Perrimon (Harvard Medical School), P. Leopold (IDV, Nizza), A. Ephrussi (EMBL Heidelberg), C. Rabouille (Hubrecht Institute), R. Cagan (Mount Sinai, NYC), E. Moreno (Universita’ di Berna), F. Schweisguth (Istituto Pasteur), M. .Gonzalez-Gaitan (Universita’ di Ginevra), B. Mellone (Universita’ del Connetticut), M. Milan (IRB, Barcellona), Andrea Ballabio (Tigem), M. Baron, Universita’ di Manchester), Hugo Bellen (Baylor College of Medicine ; Giugno 2017).

5) Attivita' Editoriale E Di Valutazione

-Il Dott. Vaccari È Editor Della Rivista Frontiers In Cell And Developmental Biology.

-Il Dott. Vaccari ha svolto la mansione di Ad hoc reviewer per le seguenti riviste scientifiche specializzate (10 articoli all’anno in media):

Nature, Nature Cell Biology, Nature communications, PLOS Biology, PLOS Genetics, PLOS One, Journal of Cell Biology, EMBO journal, EMBO reports, Current Biology, Genetics, Autophagy, Development, Journal of Cell Science, Molecular Biology of the Cell, Developmental Dynamics, Oncotargets, Physiologal Reviews, eLife, British Journal of Cancer.

-Il Dott. Vaccari ha svolto la mansione di Ad hoc reviewer per le seguenti agenzie di finanziamento (4 valutazioni all'anno in media):

European Research Council (ERC) EU; Agence Nationale Recherche (ANR) FR; Wellcome Trust UK; Association for International Cancer Research (AICR) UK; Biotechnology and Biological Sciences Research Council (BBSRC) UK

-Il Dott. Vaccari ha preso parte al comitato di valutazione di piu' di 15 tesi di dottorato per la Scuola Europea di Medicina Molecolare (SEMM) e per altre istituzioni (Istituto Pasteur, Parigi e altri):

-Il Dott. Vaccari e' membro del comitato etico di IFOM, dell' Associazione di Biologia Cellulare e del Differenziamento (ABCD) ed e' stato membro della Genetics Society of America e della Società Italiana di Biofisica e Biologia Molecolare (SIBBM).

6) Presentazioni Orali A Congressi Internazionali E Presentazioni Orali Su Invito Presso Istituzioni Scientifiche

2016 Institute of Neuropathology, Zurich, Switzerland (Host: A. Aguzzi); Unconventional Proteins and Membranes Traffic Meeting, Lecce, Italy (Organizer: G. Di Sansebastiano); 5rd JEDI Meeting, Hungary (Organizer: G. Juhasz); Italian Drosophila Meeting, Bologna, Italy (Organizers: D. Grifoni, P. Bellosta); German Drosophila Meeting, Cologne, Germany (Organizers: M. Uhlirova, A. Wodarz); 6th Conference on Targeting Notch in Cancer, Mykonos, Greece. (Organizers: A.. Epenetos, K. Brennan); Pathobiology of the Lysosome and Lysosomal Diseases, Cambridge, UK. (Organizer: J. Clarke, G. Pastores); DISS Ospedale San Paolo, Milan, Italy. (Host: V. Massa); EMBL Alumni Meeting, Rome, Italy. (Organizers: P. Avner, C. Gross); SFBD Shaping Life Meeting, Marseille, France. (Organizers: T. Lecuit, Y. Gaba, C. Maurange et al); IFOM International Scientific Meeting on chromosome replication and segregation, Milan, Italy. (Organizer: D. Branzei); CNR-Biometra, Milan, Italy. (Host: M. Venturin); Dipartimento di Bioscienze, Universita' di Milano, Milan, Italy. (Host: L. Colombo).

2015 Notch meeting, Athens, Greece (Organizers: S. Bray, S. Artavanis-Tsakonas); TIGEM, Naples, Italy (Host: A. Ballabio); EMBL-Anne Ephrussi's 60th celebration, Heidelberg, Germany (Organizer: F. Besse); IFOM/EMBL Monterotondo Symposium, Milan, Italy, (Organizers: F. di Fagagna, G. Scita); Barcelona BioMed Conference,"Drosophila as a model in cancer", Barcelona, Spain, (Organizers: C. Gonzalez, M. Milan); Nerviano Medical Science, Nerviano, Italy (Host: A. Migliazza); IFOM/MBI Joint Retreat, S. Teodoro, Italy (Organizer: S. Polo); LRI, London, UK (Host: B. Thompson), DFG Symposium "V-ATPase: A Novel Anti-Tumor Target" Milano, Italy (co-organizer with A. Vollmar); Center for Cancer Biomedicine, Norwegian Radium Hospital, Oslo, Norway (Host: T. H. Rusten); Institut Curie, Paris, France (Host: C. Thery).

2014 ABCD Meeting "Cell Biology of Disease: Cancer", Parma, Italy (Organizer: G. Scita); XVII Italian Flymeeting, Anagni, Italy (Organizer: G. Cenci); SIBBM Meeting "Emerging Arenas in Molecular Biology: from basic mechanisms to personalized medicine" Trento, Italy (Organizers: A. Cereseto, M. Denti, P. Macchi); ESF-EMBO meeting "Cell polarity and membrane traffic", Poltusk, Poland (Organizers: D. St Johnston, E. Rodriguez-Boulan); CNR for neuroscience, Milan, Italy (Host: S. Colombo); ABCD Meeting "Membrane Trafficking and Organelle Biogenesis", Pesaro, Italy (Organizer: P. Remondelli).

2013 Ludwig-Maximilian University, Munich, Germany (Host: A. Vollmar); EMBO/ FASEB Endocytosis Meeting, Villars, Switzerland (Organizer: M. Gonzalez-Gaitan); 3rd JEDI Meeting, UK (Organizer: B. Thompson); Institute Pasteur, Paris, France (Host: A. Israel); Center for Cancer Biomedicine, Norwegian Radium Hospital, Oslo, Norway (Host: H. Stenmark)

2012 ICGEB, Trieste, Italy (Host: M. Giacca); IFOM/Kyoto University Symposium, Milan, Italy
(Organizer: M. Foiani); EMBO Conference Series “Morphogenesis and Dynamics of Multicellular Systems”, EMBL, Heidelberg, Germany (Organizer: D. Gilmour/F. Peri/S. Derenzis); Beatson Institute workshop “Cold-blooded cancer. Non mammalian models for oncology research” Glasgow, UK (Organizer: M. Vidal); Gordon Conference on “Notch Signaling in Development, Regeneration &Disease”. Bates College, Maine, USA; (Organizer: R. Kopan); ESF-EMBO meeting "Cell polarity and membrane traffic", Poltusk, Poland (Organizer: C. Rabouille); DFG workshop “V-ATPase as a therapeutical target”, Munich, Germany (Organizer: A. Vollmar)

2011 University of Lausanne, Switzerland (Host: A. Mayer); EMBL, Heidelberg, Germany.
(Host: A. Ephrussi); Notch Meeting, Athens, Greece (Organizer S. Artavanis-Tsakonas); IMBC a*STAR, Singapore (Host: S. M. Cohen); Center for Cancer Biomedicine, Norwegian Radium Hospital, Oslo, Norway (Host: H. Stenmark); 1st JEDI Meeting, Leysin, Switzerland (Host: B. Deplanke, A. Stark).

2010 ETH, Zurich, Switzerland (Host: V. Panse); CIPF, Valencia, Spain (Host: S. Rodriguez-Navarro); DKFZ, Heidelberg, Germany (Host: M. Boutros); Harvard Medical School, Boston, USA (Host: T. Kirchhausen); EMBO Crete Drosophila Meeting, Crete, Greece (Organizer: C. Delidakis); University of Dusseldorf, Germany (Host: T. Klein).

2009 EMBO/FASEB Endocytosis Meeting, Crete, Greece (Organizer: H. Stenmark); European Drosophila Research Meeting, Nice, France (Organizer: S. Noselli)

2008 CRG, Barcelona, Spain (Host: V. Malhotra); LMB, Cambridge, UK (Host: M. Freeman); IRB, Barcelona, Spain (Host: M. Milan); IBDM, Marseille, France (Host: T. Lecuit); IBDC, Nice, France (Host: S. Noselli); EMBL, Heidelberg, Germany (Host: A. Ephrussi); 49th Annual Drosophila Research Conference, San Diego CA.

2006 Gordon Research Conference on ‘Lysosomes and Endocytosis’, Andover NH; 47th Annual Drosophila Research Conference, Houston TX.

2005 EMBL, Heidelberg, Germany (Host: A. Ephrussi); IFOM-IEO campus, Milan, Italy (Host: P. DiFiore/S. Sigismund); San Raffaele Institute, Milan, Italy (Host: M.E. Bianchi)

2004 CNRS/EMBO workshop ‘Epithelial Polarity in Development and Disease‘ Carry-le-Rouet, France (Organizer: T. Lecuit).

1998 Telethon Foundation Meeting, Rome, Italy.

7) Altre Partecipazioni A Congressi E Corsi

2016 EMBO workshop “Chromosome segregation and aneuploidy”. Galway, Irlanda.

2015 Telethon Foundation Meeting, Riva del Garda, Italia.

2014 ABCD Meeting on “Mechanisms of Signal Transduction”. Padova, Italia.

2013 European Drosophila Research Meeting, Barcelona, Spain.; EMBO/ESF Autophagy meeting, Bergen, Norvegia.

2012 53rd Annual Drosophila Research Conference, Chicago USA.

2011 EMBO Lab Management Course for PIs, Leiman, Germany; Notch meeting, Atene, Grecia; EMBO/FASEB Endocytosis Meeting, Crete, Grecia; European Drosophila Research Meeting, Lisbona, Portogallo; 1st JEDI Meeting, Leysin, Svizzera.

2010 EMBO meeting, Barcelona, Spain; Membrane dynamics of the cell meeting Dusseldorf, Germania; 51st Annual Drosophila Research Conference, Washington USA.

2009 Biomed Conference” Modelling Cancer in Drosophila”, Barcelona, Spain; EMBO meeting, Amsterdam, Holland; Notch meeting, Atene, Grecia; 50th Annual Drosophila Research Conference, Chicago USA.

2007 EMBO/FASEB Workshop ‘Endocytic Systems: Mechanism and Function’, Villars-sur-Ollon, Svizzera.

2005 EMBO Practical Course on ‘Endocytosis and signaling during development‘ Dresden, Germania.

2002 EMBO Practical Course on ‘Electron Microscopy, Immunocytochemistry and Stereology for Cell Biology’ EMBL, Heidelberg, Germany; EURESCO/EMBO conference 'Exocytosis Membrane Structure and Dynamics' Tomar, Portogallo.

6) Attivita’ Di Promozione Scientifica Specializzata E Non

-Il Dott. Vaccari ha co-fondato la Junior European Drosophila Investigator (JEDI) community (http://www.fly-jedi.org) e la mailing list dei Drosofilisti Italiani (italfly@googlegroups.com) e serve come consulente di Flybase (flybase.org).

Il dott. Vaccari partecipa attivamente alle attivita’ di raccolta fondi per AIRC e di divulgazione scientifica nelle scuole e per il pubblico non specializzato :
per esempio: https://www.youtube.com/watch?v=CJn1l-w0kWo https://www.youtube.com/watch?v=wtrNX1LJ_CA https://www.youtube.com/watch?v=Kznnz7cRlAw).

ATTIVITÀ DIDATTICA

1) Didattica Frontale

-Il dott. Vaccari e' Assistant Professor of Molecular Medicine per SEMM dal 2009. Nell'ambito di tale attivita' didattica insegnato nei seguenti corsi della scuola di dottorato SEMM di Oncologia Molecolare (2-3 settimane all'anno, 30-50 studenti):

2009 Cancer Genetics
2011 Animal models for research
2012 Cancer Genetics, Genetics per studenti di etica della scienza
2013 Developmental Biology

-il dott. Vaccari insegna dal 2011 "biologia cellulare e traduzione del segnale" su invito della Dott.ssa M. Beltrame del dipartimento di Bioscienze dell'Universita' di Milano, durante il corso di Biologia dello Sviluppo.

-Il dott. Vaccari ha insegnato occasionalmente e su invito "Trafficking biology, signaling and development" su invito della Dott.ssa A. Bolino per studenti di medicina dell'Universita' Vita-Salute dell'ospedale San Raffaele.

2) Altra Attivita' Didattica E Formativa

-Dal 2009 a oggi il dottor Vaccari, nella sua capacita' di capo laboratorio si e' occupato, e tuttora si occupa, di insegnare e di formare personale scientifico (per un totale di 6 tesisti, 8 dottorandi e 4 postdottorandi), tra cui attualmente:

Nome (qualifica, anno di inizio):
Marco Gualtieri (Tesista UNIMI, 2016)
Francesca Carminati (Tesista UNIMI, 2016)
Miriam Formica (Dottoranda UNIMI in collaborazione con V. Vaira/S. Bosari, 2016)
Valeria Mastrodonato (Dottoranda IFOM/SEMM, 2014)
Valentina Fajner (Dottoranda IFOM/SEMM con S. Polo, 2014)
Victor Alfred (Dottorando IFOM, 2013)
Elena Morelli (Post-dottoranda IFOM, 2011)

-In passato:

Nome (qualifica, anni di inizio e fine, posizione attuale) Informazioni sulle tesi:
-Francis Kobia (Dottorando IFOM/SEMM, 2012-2015, Postdoc Kopan lab, Cincinnati Children's hospital, USA)
Ha discusso la tesi di dottorato: "a) Pharmacologic inhibition of the vacuolar H+ ATPase reduces physiologic and oncogenic Notch signaling b) High Content Screen for Novel modulators of the Notch pathway" Esaminatore esterno: Dott. M. Baron (University of Mancherster).

-Emiliana Tognon (Dottoranda IFOM/SEMM, 2012-2015, Postdoc Longo lab, IFOM/UCSD, Italia/USA)
Ha discusso la tesi di dottorato: “Regulation of Notch signaling by the endo- lysosomal system in Drosophila: the role of ESCRT-0 and V-ATPase” Esaminatore esterno: Dott. C. Delidakis (University of Crete).

-Ilaria Busi (Tesista UNIMI Matr. n. 825829, 2013-2014, Sakura farmaceutici, Italia)
Ha discusso la tesi di laurea: “Engineering of Drosophila mutants by CRISPR/Cas9-mediated genome editing” Relatore: Dott.ssa M. Beltrame con voto finale 110/110 e lode.

-Elena Morelli (Dottoranda IFOM/SEMM, 2012-2014, Postdoc IFOM/AIRC)
Ha discusso la tesi di dottorato: “Novel functions of the SNARE protein Snap29 in membrane trafficking and cell division” Esaminatore esterno: Dott. E. Moreno (University of Bern).

-Valeria Mastrodonato (Tesista UNIMI Matr. n. 791742, 2012-2013, Dottoranda IFOM/SEMM)
Ha discusso la tesi di laurea: “Characterization of a possible role of the SNARE protein SNAP29 during cell division” Relatore: Prof.ssa E. Dejana con voto finale 110/110 e lode.

-Arianna Fumagalli (Tesista UNIMI Matr. n. 790484, 2011-2013; ora dottoranda van Rheenen lab, Hubrecht Institute, Paesi Bassi)
Ha discusso la tesi di laurea: “Developmental regulation of the vacuolar ATPase subunit c by Notch signaling in Drosophila melanogaster.” Relatore: Dott.ssa M. Beltrame con voto finale 110/110 e lode.

-Anke Witt (Tesista Hochschule Lausitz (FH) University of Applied Sciences, 2011, ora dottoranda Zerial lab, MPI-CBG Dresda, Germania)
Ha discusso la tesi di laurea: “An in vivo screen in Drosophila to uncover novel interactors of endosomal sorting in epithelial tissue” Relatore: Prof. Dr. Christian Schroeder.

-Pierpaolo Ginefra (Tesista Universita’ di Bari, 2010-2011, ora dottorando, 2010-2011 Constam lab, EPFL, Svizzera)
Ha discusso la tesi di laurea: “Clonaggio e caratterizzazione di “tumor suppressor genes in *Drosophila melanogaster*” Relatore: Prof. G. Pesole con voto finale 110/110 e lode.

Hanno inoltre frequentato il laboratorio in passato sotto la supervisione del Dott. Vaccari:
-Usha Nagarajan (Postdoc, 2012-2013; Research Fellow Queens Medical Center, University Of Nottingham, UK)
-Rohan Kadilkar (Dottorando InStem Bangalore con una EMBO short term fellowship, 2013, Postdoc Tanentzapf lab, University of British Columbia, Canada)
-Nadine Wollscheid (Dottoranda IFOM/SEMM, 2011-2012; ora Clinical Monitor IZKS, Mainz, Germania)
-Ambra Bianco (Postdoc, 2010; ora R&D Astra-Zeneca, Cambridge, UK)
-Serena Duchi (Postdoc, 2009-2011; ora Staff Scientist Istituto Ortopedico Rizzoli, Bologna).

-Dal 2009 Il Dott. Vaccari inoltre conduce lezioni per pubblico non specializzato (in media 4 all’anno) per:

scuole medie e superiori o tecniche nell’ambito dell’attivita’ raccolta fondi per AIRC e di sensibilizzazione verso la scienza (per AIRC, per il programma *Youscientist* di IFOM

https://www.youtube.com/watch?v=PCOWYqWtQK8; per la Fondazione Sodalitas, per il Collegio di Milano).

giornalisti scientifici nell'ambito dell'attivita' di aggiornamento professionale promosse da IFOM.

associazioni private su invito (Rotary Varese e altri).

PREMI E ONORIFICENZE

Nel 1997 il Dott. Vaccari consegue la laurea a pieni voti (110/110 e lode).

Nel 1999 e' vincitore di una Borsa di dottorato triennale dell'European Molecular Biology Organization (EMBO Predoctoral Fellowship).

Nel 2003 consegue il dottorato di ricerca a pieni voti (Summa cum laude).

Nel 2006 e' vincitore di una borsa biennale di postdottorato dell'American Heart Association (0625181Y).

Nel 2007 vince il premio come miglior poster all'EMBO/FASEB Workshop 'Endocytic Systems: Mechanism and Function', Villars-sur-Ollon, Svizzera

Nel 2008 e' vincitore di una borsa annuale di postdottorato dell'American Heart Association (0825176F).

Nel 2014 vince il premio come miglior poster al congresso Societa' SIBBM al congresso nazionale di Trento.

ELENCO DELLE PUBBLICAZIONI SCIENTIFICHE

1) Pubblicazioni Scientifiche In Riviste Di Lingua Inglese (Soggette A Peer Review)

Indici bibliometrici:
Citazioni totali: 2328; Citazioni medie per articolo 80; h-index: 18; Impact Factor totale: 282 (fonte Google scholar)

1. **Vaccari T**, Beltrame M, Ferrari S, Bianchi ME. (1998). Hmg4, a new member of the Hmg1/2 gene family. ***Genomics,*** 49(2), 247-52. PMID: 9598312
IF: 3.39 77 Citazioni

2. **Vaccari T**, Moroni A, Rocchi M, Gorza L, Bianchi ME, Beltrame M, DiFrancesco D. (1999). The human gene coding for HCN2, a pacemaker channel of the heart. ***Biochimica and Biophysica Acta***, 1446(3), 419-25. PMID: 10524219.
IF: 1.40 87 Citazioni

3. Golay J, Zaffaroni L, **Vaccari T**, Lazzari M, Borleri GM, Bernasconi S, Tedesco F, Rambaldi A, Introna M. (2000). Biologic response of B lymphoma cells to anti-CD20 monoclonal antibody rituximab in vitro: CD55 and CD59 regulate complement-mediated cell lysis. ***Blood***, 95(12), 3900-8. PMID: 10845926
IF: 9.27 621 Citazioni

4. Moroni A, Gorza L, Beltrame M, Gravante B, **Vaccari T**, Bianchi ME, Altomare C, Longhi R, Heurteaux C, Vitadello M, Malgaroli A, DiFrancesco D. (2001). Hyperpolarization-activated cyclic nucleotide-gated channel 1 is a molecular determinant of the cardiac pacemaker current I(f). ***Journal of Biological Chemistry***, 276(31), 29233-41. PMID: 11328811
IF: 6.48 96 Citazioni

5. **Vaccari T**, Ephrussi, A (2002). The fusome and microtubules enrich Par-1 in the oocyte, where it effects polarization in conjunction with Par-3, BicD, Egl, and dynein. ***Current Biology*** 12(17), 1524-8. PMID: 12225669.
Cover paper and F1000Prime recommendation.
IF: 11.91 45 Citazioni

6. **Vaccari T**, Rabouille C, Ephrussi, A (2005). The Drosophila PAR-1 spacer domain is required for lateral membrane association and for polarization of follicular epithelial cells. ***Current Biology*** 15(3), 255-61. PMID: 16256743.
F1000Prime recommendation
IF: 10.99 24 Citazioni

7. **Vaccari T**, Bilder D (2005).The Drosophila tumor suppressor vps25 prevents nonautonomous overproliferation by regulating notch trafficking. ***Developmental Cell*** 9(5), 687-98. PMID: 16256743.

Cover paper and F1000Prime recommendation.
IF: 13.52 300 Citazioni

8. Rusten TE, **Vaccari T**, Lindmo K, Rodahl LMW, Sem-Jacobsen C, Wendler F, Vincent JP, Brech A, Bilder D, Stenmark H (2007). ESCRTs and Fab1 regulate distinct steps of autophagy. ***Current Biology*** 17(20):1817-25. PMID: 17935992. *F1000Prime recommendation*
IF: 10.78 202 Citazioni

9. Menut L, **Vaccari T,** Dionne H, Hill J, Wu G, Bilder, D (2007). A mosaic genetic screen for Drosophila neoplastic tumor suppressor genes based on defective pupation. ***Genetics*** 177 (3):1667-77. PMID: 17947427
IF: 4.01 53 Citazioni

10. **Vaccari T**, Lu H, Kanwar R, Fortini M Bilder D (2008).Endosomal entry regulates Notch receptor activation in Drosophila melanogaster. ***Journal of Cell Biology*** 180(4): 755-62. PMID: 18299346.
F1000Prime recommendation
IF: 9.57 185 Citazioni

11. **Vaccari T***, Rusten TE, Menut L, Nesis I, Brech A, Stenmark H, Bilder D (2009). Comparative analysis of ESCRT -I, -II, -III function in Drosophila by efficient isolation of ESCRT mutants. ***Journal of Cell Science*** 122(14): 2413-2423. PMID: 1957111. *co-corresponding
IF: 6.29 99 Citazioni

12. Classen AK, Bunker BD, Harvey KF, **Vaccari T**, Bilder D (2009). A tumor suppressive activity of Drosophila Polycomb genes mediated by JAK/STAT signaling. ***Nature Genetics*** 41(10):1150-5.
PMID: 19749759
IF: 36.38 87 Citazioni

13. **Vaccari T*,** Duchi S, Cortese K, Tacchetti C, Bilder D (2010). The vacuolar ATPase is required for physiological as well as pathological activation of the Notch receptor. ***Development*** 137 (11):1825-32. PMID: 20460366.
*co-corresponding
IF: 6.90 82 Citazioni

14. Petzoldt AG, Gleixner EM, Fumagalli A, **Vaccari T*** and Simons M (2013). Activation of the proton pump, V-ATPase, triggers JNK-dependent cell invasion and overgrowth in a Drosophila epithelium. ***Disease Models & Mechanisms*** 6(3):689-700.PMID: 23335205.
* co-corresponding.
IF: 5.54 22 Citazioni

15. Kobia F, Duchi S, Deflorian G, **Vaccari T** (2014). Pharmacologic inhibition of vacuolar H+ ATPase reduces physiologic and oncogenic Notch signaling. ***Molecular Oncology*** *pii: S1574-7891(13)00160-9.* PMID: 24309677.
IF: 5.33 21 Citazioni

16. Tognon E, Wollscheid N, Cortese K, Tacchetti C, **Vaccari T** (2014). ESCRT-0 is not required for ectopic Notch activation and tumor suppression in Drosophila. ***PLoS ONE*** *9(4): e93987. doi:10.1371/journal.pone.0093987.* PMID: 24718108.

IF: 3.23 8 Citazioni

17. Morelli E, Ginefra P, Mastrodonato V, Beznoussenko G, Rusten TE, Bilder D, Stenmark H, Mironov AA, **Vaccari T** (2014). Multiple functions of the SNARE protein Snap29 in autophagy, endocytic and exocytic trafficking during epithelial formation in Drosophila. ***Autophagy*** *10(12):2251-68. doi:10.4161/15548627.2014.981913.* PMID: 25551675.
IF: 11.75 10 Citazioni

18. Handschuh K, Feenstra J, Koss M, Ferretti E, Risolino M, Zewdu R, Sahai MA, Bénazet JD, Peng XP, Depew MJ, Quintana L, Sharpe J, Wang B, Alcorn H, Rivi R, Butcher S, Manak JR, **Vaccari T,** Weinstein H, Anderson KV, Lacy E, Selleri L. (2014). ESCRT-II/Vps25 constrains digit number by endosome-mediated selective modulation of FGF-SHH signaling. ***Cell Reports*** *9(2):674-87. doi: 10.1016/j.celrep.2014.09.019.* PMID: 25373905.
IF: 7.20 6 Citazioni

19. Di Cristofori A, Ferrero S, Bertolini I, Gaudioso G, Russo MV, Berno V, Vanini M, Locatelli M, Zavanone M, Rampini P, **Vaccari T**, Caroli M, Vaira V (2015). The vacuolar H+ ATPase is a novel promising therapeutic target for glioblastoma. ***Oncotarget*** *6(19): 17514-31.* PMID: 26020805
IF: 6.36 8 Citazioni

20. Tognon E, Kobia F, Busi I, Fumagalli A, De Masi F, **Vaccari T** (2016). Control of lysosomal biogenesis and Notch-dependent tissue patterning by components of the TFEB/ V-ATPase axis in Drosophila melanogaster. ***Autophagy*** *12(3):499-514. doi: 10.1080/15548627.2015.1134080. PMID: 26727288*
IF: 11.75 4 Citazioni

21. Morelli E, Mastrodonato V, Beznoussenko G, Mironov AA, Tognon E, **Vaccari T** (2016). An essential step of kinetochore formation controlled by the SNARE protein Snap29. ***EMBO Journal*** *35:2223-2237, doi: 10.15252/embj.201693991*
IF: 10.43

2) Reviews, Articoli Tecnici, Commenti E Capitoli Di Libri Su Riviste In Lingua Inglese (Soggette A Peer Review)

1. **Vaccari T***, Bilder D (2009). At the crossroads of polarity, proliferation and apoptosis: The use of Drosophila to unravel the multifaceted role of endocytosis in tumor suppression. ***Molecular Oncology*** 3(4): 354-365. Invited review on thematic issue: “Endocytosis, Signaling And Cancer, Much More Than Meets The Eye”. PMID: 19560990. *co-corresponding
IF: 4.53 38 Citazioni

2. Rusten TE, **Vaccari T**, Stenmark H (2011). Shaping Development with ESCRTs. ***Nature Cell Biology***. 22;14(1):38-45. PMID: 22193162
IF: 20.06 86 Citazioni

3. Thompson B, Perez F, **Vaccari T** (2012). The young and happy marriage of membrane traffic and cell polarity. ***EMBO reports***. 13(8):670-2. PMID: 22777496
IF: 7.86 1 Citazioni

4. Boniolo G, **Vaccari T** (2012). Publishing: Alarming shift away from sharing results. ***Nature****. (Correspondence).* 9;488(7410):157. PMID: 22874955
F: 42.35 2 Citations

5. Tognon E, **Vaccari T** (2014). Immuno-histochemical tools and techniques to visualize Notch in Drosophila melanogaster. ***Methods in Molecular Biology.*** *1187: 63-78. Editore: Hugo J. Bellen.*PMID: 25053481
IF: 1.29 1 Citazione

6. Cuomo A, Sanfilippo R, **Vaccari T,** Bonaldi T (2014). Proteomics meets genetics: SILAC labeling of Drosophila melanogaster larvae and cells for in vivo functional studies. ***Methods in Molecular Biology.*** *1188: 293-311. Editore: Bettina Warscheid.* PMID: 25059620.
IF: 1.29 1 Citazione

7. Klionsky D, ..., **Vaccari T** , e altri.. (2016). Guidelines for the Use and Interpretation of Assays for Monitoring Autophagy (3rd edition). ***Autophagy****, Jan 2;12(1):1-222.* PMID: 26799652
IF: 11.75 163 Citazioni

8. Alfred V, **Vaccari T** (2016). When membranes need an ESCRT: Endosomal sorting and membrane remodeling in health and disease. *Invited review by A. Aguzzi for* ***Swiss Medical Weekly*** 146:w14347. PMID: 27631343

9. Mastrodonato V, Morelli E, **Vaccari T** (2016). Autophagy in non-mammalian systems (version 2.0). *Invited book chapter by G. Melino for* ***eLS - encyclopedia of Life Sciences,*** *doi: 10.1002/9780470015902.a0021582.pub2*

3) Comunicazioni A Congresso In Forma Di Abstract (Non Soggette A Peer Review)

La lista e' parziale. Si riferisce solo al periodo del Dott. Vaccari come capo laboratorio (2009-2016) e non include gli abstract selezionati per una comunicazione orale.

1. **Vaccari T,** Bilder D Vacuolar Atpase Activity Controls Physiologic As Well As Pathologic Notch Signaling Activation In Drosophila. 2009 Biomed Conference" Modelling Cancer In Drosophila", BarceLLona, Spagna.

2. Duchi S, Cortese K, Tacchetti C, Bilder D, **Vaccari T**. Analisi Dell'attivazione Mediata Da Endocitosi Del Recettore Notch. 2010 Italian Flymeeting, Lecce, Italia.

3. Duchi S, Cortese K, Tacchetti C, Bilder D, **Vaccari T.** Vacuolar Atpase, A Regulated And Conserved Component Of The Notch Pathway. 2011, Notch Meeting Atene, Grecia.

4. Duchi S, Cortese K, Tacchetti C, Bilder D, **Vaccari T.** The Role Of V-Atpase In Notch Signaling. 2011, Abcd Meeting, Ravenna.

5. Morelli E, Ginefra P, **Vaccari T**. Characterization Of The Snare Protein Snap29 In Trafficking, Signaling And Tumor Suppression. 2011 Abcd Meeting, Ravenna.
6. Woessner (Wollscheid) N, Wang X, Montell Dj, **Vaccari T.** Escrt-0 Controls Jak/Stat Signaling And Tumor Suppression In Drosophila 2011 Abcd Meeting, Ravenna.

7. Woessner (Wollscheid) N, Wang X, Montell Dj, **Vaccari T.** Escrt-0 Controls Jak/Stat Signaling And Tumor Suppression In Drosophila 2011 European Drosophila Conference Lisbona, Portogallo.

8. Morelli E, Ginefra P, **Vaccari T**. Characterization Of Snap29 In Trafficking, Signaling And Tumor Suppression 2011 European Drosophila Conference Lisbona, Portogallo.

9. Morelli E, Ginefra P, Bilder, D **Vaccari T**. Characterization Of Snap29 In Trafficking, Signaling And Tumor Suppression 2012 Italian Flymeeting Palermo, Italia.

10. Kobia F, Duchi S, **Vaccari T.** Reduction Of Notch Signaling By Pharmacologic Inhibition Of V-Atpase Activity. 2012 Italian Flymeeting Palermo, Italia.

11. Fumagalli A, Tognon E, Duchi S, **Vaccari T.** Regulation Of The V-Atpase Expression During Developmental Signaling. 2012. Italian Flymeeting Palermo, Italia.

12. Morelli E, Ginefra P, Bilder, D **Vaccari T**. The Snare Protein Snap29, Which Is Compromised In Cednik Syndrome, Is A Novel Regulator Of Autophagosome Maturation. 2013 Embo Workshop On Autophagy Bergen Norway.

13. Witt A, Alfred V, Nagarajan U, **Vaccari T.** A Deficiency Screen To Identify Genetic Interactors Of Escrt-Ii Subunit, Vps25 In Drosophila Melanogaster 2013 Abcd Meeting Ravenna, Italy.
14. Kobia F, Duchi S, Deflorian G, **Vaccari T**. Pharmacologic Inhibition Of Vacuolar H+ Atpase Reduces Physiologic And Oncogenic Notch Signaling. 2013 Abcd Meeting Ravenna, Italy.
15. Tognon E, Fumagalli A, **Vaccari T**. Regulation Of The V-Atpase Expression During Developmental Signaling. 2013 Abcd Meeting Ravenna, Italy.
16. Morelli E, Ginefra P, Mastrodonato V, Beznoussenko G, Mironov A, Sprecher E, Ishida-Yamamoto A, Rusten T, Stenmark H, Bilder D, **Vaccari T** The Snare Protein Snap29 Controls Formation Of Epithelial Organs And Autophagy In Drosophila 2013 Abcd Meeting Ravenna, Italla.

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