

Curriculum vitae

Michela Clerici

Personal data

Nationality	Italian
Birth	February 27th, 1975 in Bollate (MI), Italy
Current position	Associate Professor in Genetics (SSD BIO18) at the University of Milano-Bicocca
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Author profile

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ERC panel

LS1_3 DNA and RNA biology

Bibliometric indicators

H-index (Scopus source)	23
Total peer-reviewed publications (in journals with IF)	35
Total number of citations (Scopus source)	2076

The results of Michela Clerici's research activity are documented by 35 papers, which have been published in international peer-reviewed journals with Impact Factors, and two book chapters. She is corresponding author of nine of these papers.

Professional career

March 2015-present	Associate Professor in Genetics at the Department of Biotechnology and Biosciences, University of Milano-Bicocca
2008-2015	Assistant Professor in Genetics at the Department of Biotechnology and Biosciences, University of Milano-Bicocca
2004-2008	Post-doctoral fellow in the laboratory of Prof. Maria Pia Longhese at the Department of Biotechnology and Biosciences, University of Milano-Bicocca (fellowships from University of Milano-Bicocca and Fondazione Telethon)

2000-2003	PhD student in the laboratory of Prof. Maria Pia Longhese at the University of Milano-Bicocca
2000	Graduate fellow in the laboratory of Dr. Maria Pia Longhese (fellowship from “Biopolo”)
1999-2000	Undergraduate student in the laboratory of Prof. Giovanna Lucchini and Dr. Maria Pia Longhese at the Department of Genetics, University of Milano.

Education

Gen. 2004	PhD in Industrial Biotechnology at the University of Milano-Bicocca
July 2000	Degree in Biology, Summa cum Laude, at the University of Milano

Technical skills and competences

Expert in yeast genetics, Michela Clerici is experienced with genetic, biochemistry, molecular biology and cell biology techniques, as well as with fluorescence microscopy and image processing. She is particularly skilled in generating mutants by both site-specific and random mutagenesis and in performing genome-wide genetic screenings. Finally, she contributed to generate a wide collection of yeast strains and mutants.

Scientific activity

Michela Clerici has always been using the budding yeast *Saccharomyces cerevisiae* as a eukaryotic model system to explore the mechanisms preserving genome stability in eukaryotes, focusing in particular on the cellular response to DNA damage and replication stress, as well as on the control of the homeostasis of telomeres, the nucleoprotein specialized structures that protect the natural ends of chromosomes. Genetic instability can induce tumorigenesis in multicellular organisms, and replication stress is emerging as a hallmark of cancer cells. Therefore, unveil the molecular mechanisms underlying the response to DNA perturbations is of considerable biological relevance.

The cellular response to DNA damage and replication stress is orchestrated by evolutionarily conserved checkpoint pathways, which preserve genome stability by delaying cell cycle progression in the presence of DNA alterations, concomitantly activating DNA repair systems and regulating DNA replication. Thanks to the use of multidisciplinary approaches, Michela Clerici’s research has provided significant insights into these mechanisms. In particular she contributed to elucidate i) the functions and regulation of different checkpoint proteins, among which the central conserved protein kinases Mec1/ATR and Tel1/ATM, ii) the complex relationships among checkpoint and DNA repair systems, iii) the role of the master regulator of cell cycle progression, the cyclin-dependent kinase Cdk1, in promoting specific homologous recombination-dependent repair pathways, and iv) the molecular mechanisms regulating the nucleolytic processing of DNA ends, an event particularly important to repair DNA double-strand breaks and to maintain telomere stability.

Her recent research activity has been unveiling a novel and specific function of the checkpoint kinase Tel1/ATM in supporting DNA replication upon the generation of a topological stress imposed by the poisoning of DNA topoisomerase, and the role of chromatin remodeling factors in promoting DNA repair.

Academic activity

Since 2001 Michela Clerici has been supervise several undergraduate and Ph.D students at the University of Milano-Bicocca.

Teaching activities at the University of Milano-Bicocca:

2022-present “Molecular and cellular oncology”, Master’s degree course in Biology (42 hours)

2015-2021 “Analysis of gene functions”, Bachelor's degree course in Biotechnology (42 hours)

2013-2014 “Analysis of gene functions”, Bachelor's degree course in Biotechnology (21 hours)

2008-present “Laboratory of genetics technologies”-Practical course, Bachelor's degree course in Biotechnology (90 hours)

Research grants and Awards

- 2013-2021 Finanziamenti Progetti di Ateneo-Università di Miano-Bicocca
- 2019 Competitive grant-Università degli studi di Milano-Bicocca “Exploiting the replication stress response to tackle MYC-driven tumors” (15.000 €)
- 2010-2013 MIUR-PRIN2009 “Exploring the link between gene transcription and telomere regulation in *Saccharomyces cerevisiae*”. (63.000 €)
- 2007 Giovanni Magni award from Adriano-Buzzati Traverso Foundation for the best paper published by a young researcher in microorganism genetics (The *S. cerevisiae* Sae2 protein negatively regulates DNA damage checkpoint signaling. 2006. *EMBO Reports*, 7:212-218).

Publications in the last 10 years

1. Frigerio C, Di Nisio E, Galli M, Colombo CV, Negri R, Clerici M. 2023. The chromatin landscape around DNA double-strand breaks in yeast and its influence on DNA repair pathway choice. *International Journal of molecular sciences*, 24, 3248-3271. doi: 10.3390/ijms24043248.
2. Casari E, Gobbini E, Gnugnoli M, Mangiagalli M, Clerici M, Longhese MP. 2021. Dpb4 promotes resection of DNA double-strand breaks and checkpoint activation by acting in two different protein complexes. *Nat Commun.* 12:4750. doi: 10.1038/s41467-021-25090-9.
3. Bonetti D, Clerici M, Longhese MP. 2021. Interplay between Sae2 and Rif2 in the regulation of Mre11-Rad50 activities at DNA ends. *Curr Opin Genet Dev.* 71:72-77. doi: 10.1016/j.gde.2021.07.001.
4. Galli M, Frigerio C, Longhese MP, Clerici M. 2021. The regulation of the DNA damage response at telomeres: focus on kinases. *Biochem Soc Trans.* 49:933-943. doi: 10.1042/BST20200856.
5. Casari E, Gobbini E, Clerici M, Longhese MP. 2021. Resection of a DNA Double-Strand Break by Alkaline Gel Electrophoresis and Southern Blotting. *Methods Mol Biol.* 2153:33-45. doi: 10.1007/978-1-0716-0644-5_3.
6. Menin L, Colombo CV, Maestrini G, Longhese MP, Clerici M. 2019. Tel1/ATM Signaling to the Checkpoint Contributes to Replicative Senescence in the Absence of Telomerase. *Genetics.* 213:411-429. doi: 10.1534/genetics.119.302391.

7. Colombo CV, Menin L, Ranieri R, Bonetti D, Clerici M, Longhese MP. 2019. Uncoupling Sae2 Functions in Downregulation of Tel1 and Rad53 Signaling Activities. *Genetics*. 211:515-530. doi: 10.1534/genetics.118.301830.
8. Menin L, Ursich S, Trovesi C, Zellweger R, Lopes M, Longhese MP and Clerici M. 2018. Tel1/ATM prevents degradation of replication forks that reverse after topoisomerase poisoning. In stampa in *EMBO reports*
9. Colombo CV, Menin L, Clerici M. 2018. Alkaline Denaturing Southern Blot Analysis to Monitor Double-Strand Break Processing. *Methods Mol Biol*. 1672:131-145. doi: 10.1007/978-1-4939-7306-4_11.
10. Colombo CV, Trovesi C, Menin L, Longhese MP, Clerici M. 2017. The RNA binding protein Npl3 promotes resection of DNA double-strand breaks by regulating the levels of Exo1. *Nucleic Acids Res*. 45:6530-6545. doi: 10.1093/nar/gkx347.
11. Cassani C, Gobbini E, Wang W, Niu H, Clerici M, Sung P, Longhese MP. 2016. Tel1 and Rif2 Regulate MRX Functions in End-Tethering and Repair of DNA Double-Strand Breaks. *PLoS Biol*. 14:e1002387. doi: 10.1371/journal.pbio.1002387.
12. Gobbini E, Villa M, Gnugnoli M, Menin L, Clerici M, Longhese MP. 2015. Sae2 Function at DNA Double-Strand Breaks Is Bypassed by Dampening Tel1 or Rad53 Activity. *PLoS Genet*. 11:e1005685. doi: 10.1371/journal.pgen.1005685.
13. Manfrini N, Clerici M, Wery M, Colombo CV, Describes M, Morillon A, d'Adda di Fagagna F, Longhese MP. 2015. Resection is responsible for loss of transcription around a double-strand break in *Saccharomyces cerevisiae*. *Elife*. 4. doi: 10.7554/eLife.08942.
14. Clerici M, Trovesi C, Galbiati A, Lucchini G, Longhese MP. 2014. Mec1/ATR regulates the generation of single-stranded DNA that attenuates Tel1/ATM signaling at DNA ends. *EMBO J*. 33:198-216. doi: 10.1002/emboj.201386041.
15. Bonetti D, Anbalagan S, Lucchini G, Clerici M, Longhese MP. 2013. Tbf1 and Vid22 promote resection and non-homologous end joining of DNA double-strand break ends. *EMBO J*. 32:275-89. doi: 10.1038/emboj.2012.327.
16. Fumagalli M, Rossiello F, Clerici M, Barozzi S, Cittaro D, Kaplunov JM, Bucci G, Dobrev M, Matti V, Beausejour CM, Herbig U, Longhese MP, d'Adda di Fagagna F. 2012. Telomeric DNA damage is irreparable and causes persistent DNA-damage-response activation. *Nat Cell Biol*. 14:355-65. doi: 10.1038/ncb2466.
17. Martina M, Clerici M, Baldo V, Bonetti D, Lucchini G, Longhese MP. 2012. A balance between Tel1 and Rif2 activities regulates nucleolytic processing and elongation at telomeres. *Mol Cell Biol*. 32:1604-17. doi: 10.1128/MCB.06547-11.

Selected publications before 2012

1. Trovesi C, Falcettoni M, Lucchini G, Clerici M, Longhese MP. 2011. Distinct Cdk1 requirements during single-strand annealing, noncrossover, and crossover recombination. *PLoS Genet*. 2011 7:e1002263. doi: 10.1371/journal.pgen.1002263.
2. Bonetti D, Martina M, Clerici M, Lucchini G, Longhese MP. 2009. Multiple pathways regulate 3' overhang generation at *S. cerevisiae* telomeres. *Mol Cell*. 35:70-81. doi: 10.1016/j.molcel.2009.05.015.
3. Clerici M, Mantiero D, Guerini I, Lucchini G, Longhese MP. 2008. The Yku70-Yku80 complex contributes to regulate double-strand break processing and checkpoint activation during the cell cycle. *EMBO Rep*. 9:810-8. doi: 10.1038/emboj.2008.121.

4. Mantiero D*, Clerici M*, Lucchini G, Longhese MP. 2007. Dual role for *Saccharomyces cerevisiae* Tel1 in the checkpoint response to double-strand breaks. *EMBO Rep.* 8(4):380-7.
5. Clerici M, Mantiero D, Lucchini G, Longhese MP. 2006. The *Saccharomyces cerevisiae* Sae2 protein negatively regulates DNA damage checkpoint signalling. *EMBO Rep.* 7:212-8.
6. Clerici M, Mantiero D, Lucchini G, Longhese MP. 2005. The *Saccharomyces cerevisiae* Sae2 protein promotes resection and bridging of double strand break ends. *J Biol Chem.* 280:38631-8.
7. Clerici M, Baldo V, Mantiero D, Lottersberger F, Lucchini G, Longhese MP. 2004. A Tel1/MRX-dependent checkpoint inhibits the metaphase-to-anaphase transition after UV irradiation in the absence of Mec1. *Mol Cell Biol.* 24:10126-44.
8. Clerici M, Paciotti V, Baldo V, Romano M, Lucchini G, Longhese MP. 2001. Hyperactivation of the yeast DNA damage checkpoint by TEL1 and DDC2 overexpression. *EMBO J.* 20:6485-98.
9. Paciotti V*, Clerici M*, Scotti M, Lucchini G, Longhese MP. 2001. Characterization of mec1 kinase-deficient mutants and of new hypomorphic mec1 alleles impairing subsets of the DNA damage response pathway. *Mol Cell Biol.* 21:3913-25.
10. Paciotti V, Clerici M, Lucchini G, Longhese MP. 2000. The checkpoint protein Ddc2, functionally related to *S. pombe* Rad26, interacts with Mec1 and is regulated by Mec1-dependent phosphorylation in budding yeast. *Genes Dev.* 14:2046-59.

*Equal contribution to this work

Milano, 23/02/2023

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