BIOGRAPHICAL SKETCH

NAME: CRISTINA LANNI

eRA COMMONS USER NAME (credential, e.g., agency login): CRISTINA_LANNI

POSITION TITLE: Associate Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Start Date MM/YYYY	Completion Date MM/YYYY	FIELD OF STUDY
Classical studies high school diploma, Liceo Classico San Giorgio, Pavia, Italy	Baccalaureate	09/1990	07/1995	
University of Pavia, School of Biological Sciences, Pavia, Italy	B.S.	10/1995	07/2000	Physiopathology
University of Pavia, Board Certification in Applied Pharmacology (medical area), Pavia, Italy	M.S.	09/2001	07/2005	Neuropharmacology
University of Pavia, International Doctorate in Biomolecular Sciences and Biotechnology, Pavia, Italy	Ph.D.	11/2005	12/2008	Neuropharmacology
Scuola Avanzata di Formazione Integrata" (SAFI) of the Scuola Superiore IUSS, Pavia, Italy	Higher Post Graduate Education	01/2003	06/2006	CNS disorders

A. Personal Statement

Cristina Lanni has a great expertise in the field of neurodegeneration and age-related disorders. At the beginning of her scientific career, she was initially oriented on the pathogenic mechanisms of Alzheimer's disease (AD), and in particular on the pharmacological regulation of amyloid precursor protein metabolism and on the neurotoxicity of beta-amyloid peptide (Aβ). Based on her background she took also part to different scientific collaborations, contributing in the scientific research with data on a dual role of Aβ strictly correlated with its concentration (neuromodulatory/neuroprotective vs neurotoxic). In particular she focused on the molecular characterization of the hypothetical physiological effect of Aβ on cellular network, by examining the physiological effects of Aβ on acute synaptic activities and the functional interplay existing between Aβ and different neurotransmitter systems. In understanding the etiopathogenesis of AD, Cristina Lanni has also participated to research new potential peripheral biomarkers for Alzheimer's disease, by characterizing and describing the modulation of the conformational state of p53 by physiological concentrations of Aβ. At the moment, Cristina Lanni is researching on the bidirectional link between gastrointestinal inflammation and neurodegeneration, with particular attention to circadian clockwork modulation in the periphery and in the brain.

***Research support**:*

- Funding "Young Investigator Award" at University of Pavia for the project "Lithium in Alzheimer's disease: protection against beta-amyloid-induced neurodegeneration" Goal: Characterization of lithium ability to rescue from beta-amyloid toxicity (12 months). Role: PI
- Grant PRIN 2007HJCCSF_001 for the research "Beta amyloid, the culprit of Alzheimer's neurodegeneration or a new player in brain physiological and pathological neuromodulation?" Goal: Evaluation of the in vitro effect of beta amyloid on dopamine release and on signal transduction machinery downstream presynaptic receptors controlling neurotransmitter release (24 months). Role: Partecipant.
- Funding "Progetto Regione Lombardia - Almamater" SAL45-17261, for the research "From materials sciences to development of new devices for the diagnosis and cure of aging related disorders" Goal: Dissection of the effect of Aβ on p53 structure and functions (24 months). Role: Co-PI.
- Grant PRIN 2020SCBBN2_005 for the research "Glymphatic system: a new player in the gut-brain axis. Natural resources to maintain homeostasis". Goal: To evaluate the relationship between gut dysbiosis and glymphatic system alteration and to characterize the healthy properties of a food supplement based on plant extracts for relieving gut disequilibrium and the associated disorders focusing on brain dysfunctions due to a deficient CSF diffusion and waste clearance (36 months). Role: Co-PI.

- National Grant: F13C22001110001_ IMMUNO-HUB Ministero della Salute – Piano Operativo Salute, PSC. Traiettoria 4 – Interventi per la creazione di Hub delle Scienze della Vita nei settori della Farmaceutica, del Biomedicale e delle Biotecnologie (36 months). Role: Partecipant.
- Grant AARG-23-1140660 for the research «Misalignment in circadian glymphatic system as trigger of neurodegeneration» Goal: To establish the threshold (or time window) beyond which the glymphatic system impairment and pathological waste deposition, due to circadian misalignment, becomes irreversible and triggers neurodegenerative processes (36 months). Role: PI

B. Positions, Scientific Appointments and Honors

Professional Experience:
- 2023, September: Qualification to the role of Full Professor in Pharmacology
- 2015, 1st August: Associate Professor in Pharmacology, University of Pavia
- 2008, 29th December: Assistant Professor in Pharmacology, University of Pavia
- 2005-2008: Biomolecular Sciences and Biotechnology Doctorate fellowship, University of Pavia, on the project "Alzheimer's disease, new diagnostic and therapeutic tools: focus on p53".
- 2004-2005: Recipient of fellowship, Department of Experimental and Applied Pharmacology, University of Pavia to the study of beta-amyloid peptide as therapeutic target in Alzheimer's disease.
- 2003-2004: Recipient of fellowship, "Istituto Neurologico Casimiro Mondino", Pavia, to the study of biological markers in Alzheimer's disease.
- 2001-2003: Recipient of fellowship, Department of Experimental and Applied Pharmacology, University of Pavia to the study of the role of beta-amyloid peptide.

Honors and Awards:
2002 Neuropsycopharmacology Italian Society (SINPF) Poster Award for researches on the neuroprotection in Alzheimer's Disease
2003 Celltox (Associazione Italiana Tossicologia in vitro) Travel Award
2004 SAFI (Scuola Avanzata di Formazione Integrata) Study Award on CNS pathologies
2005 SAFI (Scuola Avanzata di Formazione Integrata) Study Award on CNS pathologies
2009 12th International Conference on Alzheimer's Disease (ICAD) Travel Fellowship
2019 PI_Internal Quota ricerca FRG2019, Department of Drug Science, Pavia
2020 PI_Internal Quota ricerca FRG2020, Department of Drug Science, Pavia
2020 Outstanding Reviewer Award - Certificate of Achievement Signal Transduction and Targeted Therapy (SpringerNature)

Memberships in Scientific Societies
Cristina Lanni is a member of Alzheimer's Association International Society to Advance Alzheimer's Research and Treatment (ISTAART), Italian Society for Pharmacology, Italian Society for Neuroscience and Italian Society of Neuropsychopharmacology. In addition, Cristina Lanni is included in the board of AMAE (Associazione Malati Acalasia Esofagea) Onlus (www.amae.it) and to date she is the chairman of the scientific committee of this association, whose activities are focused on increasing awareness for the rare disease achalasia.

Editorial Board
Associate Editor for Neurodegeneration - Frontiers in Neuroscience; Frontiers Topic Editor – Immune response modulation by nanoparticle formulations; Guest Associate Editor in Pharmacology of Anti-Cancer Drugs; Review Editor for Neurocognitive Aging and Behavior - Frontiers in Aging Neuroscience; Review Editor for Geriatric Medicine - Frontiers in Medicine
Reviewer for the following scientific journals: Signal Transduction and Targeted Therapy, Molecular Neurobiology, Scientific Reports, Frontiers in Pharmacology Experimental Pharmacology and Drug Discovery, Frontiers in Molecular Biosciences Molecular Diagnostics and Therapeutics, Journal of Neuroscience Methods, Brain Research Bulletin, Neurotoxicity Research, International Journal of Alzheimer's disease, Plos One, Pharmacological Research, Biology.

C. Contributions to Science

The contributions to science are detailed below with the most significant references from the complete list of publications. They can be divided into:
I. Identification of peripheral markers of Alzheimer's disease
II. Pharmacological regulation of amyloid precursor protein (APP) metabolism and on the possible neuromodulatory role of the beta amyloid peptide
III. Neurotoxic activity of beta amyloid and approaches targeting beta-amyloid for therapeutic intervention of Alzheimer's disease
IV. Glymphatic system: a new view in the gut-brain axis

I - Identification of peripheral markers of Alzheimer's disease

Early diagnosis of Alzheimer's disease (AD) is a crucial starting point in disease management. Blood-based biomarkers could represent an advantage in providing AD-risk information in primary care settings, and in monitoring the effectiveness of any therapies. Candidate biochemical markers for AD should be molecules that represent some of the brain pathogenetic processes typical of the disease or alterations in metabolic or cellular processes, as demonstrated by numerous studies performed on both the brain and peripheral tissues of affected patients. Cristina Lanni found that fibroblasts and peripheral blood cells from sporadic AD patients specifically express an anomalous and detectable conformational state of p53 that makes these cells distinguishable from cells of age-matched non-AD subjects, thus supporting the existence of a putative marker for AD [1]. When investigating the mechanism of such alteration, she also demonstrated that the exposure to nanomolar concentrations of beta-amyloid induces the expression of unfolded p53 protein isoform in different AD models, by affecting the expression and activity of different proteins regulating p53 function [2]. In particular, Cristina Lanni provided evidence that soluble non-toxic beta amyloid is involved in disrupting the physiological functions of p53 producing a cellular phenotype with an aberrant control of cell death pathways eventually resulting in the survival of injured dysfunctional cells. As an extension of this research, Cristina Lanni developed and applied in an original way immunocytochemical and flow cytometry techniques to identify conformationally altered p53 in blood cells deriving from AD and by subjects with mild cognitive impairment [3]. Based on these data, to date, the AlzoSure® Predict test has been developed to quantify the AZ 284® peptide as readout of the unfolded conformational variant of p53 (U-p53AZ) [4].

This research has been supported by: Funding "Progetto Regione Lombardia - Almamater" SAL45-17261, for the project "From materials sciences to development of new devices for the diagnosis and cure of aging related disorders" Goal: Dissection of the effect of Aβ on p53 structure and functions (24 months). Role: Co-PI.

References

[1] Lanni C, Racchi M, Mazzini G, Ranzenigo A, Polotti R, Sinforiani E, Olivari L, Barcikowska M, Styczynska M, Kuznicki J, Szybinska A, Govoni S, Memo M, Uberti D. Mol Psychiatry. 2008;13(6):641-7.

[2] Lanni C, Necchi D, Pinto A, Buoso E, Buizza L, Memo M, Uberti D, Govoni S, Racchi M. J Neurochem. 2013;125(5):790-9.

[3] Lanni C, Racchi M, Stanga S, Mazzini G, Ranzenigo A, Polotti R, Memo M, Govoni S, Uberti D. J Alzheimers Dis. 2010;20(1):97-104.

[4] Piccirella S, Van Neste L, Fowler C, Masters CL, Fripp J, Doecke JD, Xiong C, Uberti D, Kinnon P. J Prev Alzheimers Dis. 2022;9(3):469-479.

II - Pharmacological regulation of amyloid precursor protein (APP) metabolism and on the possible neuromodulatory role of the beta amyloid peptide

The research conducted by Cristina Lanni was oriented to characterize the intracellular transduction pathways modulating the non-amyloidogenic metabolism of APP, thus leading to the release of soluble APP and the reduction in beta amyloid production. In particular, Cristina Lanni directly contributed to dissect the function of phosphorylations regulated by protein kinase C (PKC), as well as by casein kinase 2 (CK2), defining the specific roles for different isoforms of PCK and for CK2 in the pharmacological modulation of APP metabolism [1-2]. Furthermore, Cristina Lanni also contributed in defining the functions and the mechanisms of APP degradation regulating the release of the amyloid precursor protein intracellular domain, known as AICD.

When focusing on beta-amyloid, besides its widely investigated role as the main pathogenic marker responsible for neurodegeneration in AD, Cristina Lanni participated to a strong research line indicating β-amyloid (Aβ) as a normal product of neuronal metabolism, acting as a crucial regulator of key physiological functions at synapse. In particular, she contributed to demonstrate that Aβ regulates the release of several neurotransmitters, including dopamine, GABA, glutamate, aspartate, and glycine, by mainly affecting the cholinergic control of their release, in conditions not resulting in neurotoxicity [3-4].

This research has been supported by: PRIN 2007HJCCSF_001 for the project "Beta amyloid, the culprit of Alzheimer's neurodegeneration or a new player in brain physiological and pathological neuromodulation?" Goal: Evaluation of the in vitro effect of beta amyloid on dopamine release and on signal transduction machinery downstream presynaptic receptors controlling neurotransmitter release (24 months). Role: Partecipant.

References

[1] Lanni C, Mazzucchelli M, Porrello E, Govoni S, Racchi M. Eur J Biochem. 2004;271(14):3068-75.

[2] Lenzken SC, Stanga S, Lanni C, De Leonardis F, Govoni S, Racchi M. J Alzheimers Dis. 2010;20(4):1133-41.

[3] Mura E*, Lanni C*, Preda S, Pistoia F, Sarà M, Racchi M, Schettini G, Marchi M, Govoni S. Curr Pharm Des. 2010;16(6):672-83. * equal contribution

[4] Mura E, Zappettini S, Preda S, Biundo F, Lanni C, Grilli M, Cavallero A, Olivero G, Salamone A, Govoni S, Marchi M. PLoS One. 2012;7(1):e29661.

III - Neurotoxic activity of beta amyloid and approaches targeting beta-amyloid for therapeutic intervention of Alzheimer's disease

Most AD drugs currently available can only alleviate symptoms rather than modify the underlying molecular cause of AD. In the field of therapeutic interventions targeting at various beta-amyloid (Aβ)-associated pathological mechanisms of AD,

Cristina Lanni contributed in better characterizing the kinetics and the chemical and physical factors contributing the Aβ nucleation from monomer to fibrils. In particular, she participated in striking differences in the electrophoretic patterns of Aβ 1-42 and Aβ 1-40 over time, and in elucidating different aggregation states, which reflect the diverse oligomerization behavior of two very similar peptides as well as the different profile of toxicity [1]. Based on this characterization, Cristina Lanni then contributed in the design and characterization of the mechanism of action of different synthetic and hybrid compounds able to interfere with the neurotoxic properties of Aβ [2-4]. This research led to a national patent C.T.I.N. 0001387037 on "Derivati antracenedionici e aza-antracenedionici come agenti capaci di inibire l'aggregazione di peptidi beta-amiloidi", in which Cristina Lanni is co-inventor.

References

[1] Bisceglia F, Natalello A, Serafini MM, Colombo R, Verga L, Lanni C, De Lorenzi E. Talanta. 2018;188:17-26.
[2] Colombo R, Carotti A, Catto M, Racchi M, Lanni C, Verga L, Caccialanza G, De Lorenzi E. Electrophoresis. 2009;30(8):1418-29.
[3] Simoni E, Serafini MM, Bartolini M, Caporaso R, Pinto A, Necchi D, Fiori J, Andrisano V, Minarini A, Lanni C*, Rosini M*. ChemMedChem. 2016;11(12):1309-17. * equal contribution
[4] Serafini MM, Catanzaro M, Fagiani F, Simoni E, Caporaso R, Dacrema M, Romanoni I, Govoni S, Racchi M, Daglia M, Rosini M, Lanni C. Front Pharmacol. 2020;10:1597.

IV – Glymphatic system: a new view in the gut-brain axis.

Increasing evidence supports the reciprocal communication between the enteric and the central nervous system (CNS) in disease, with a critical role of peripheral inflammatory processes in the pathogenesis of dementia. Cristina Lanni contributed to this field by investigating the role of the dysfunctionality of the glymphatic system (a fluid-clearance pathway that consists of periarterial cerebro-spinal fluid inflow running in the same direction as blood flow, ensuring the clearance of metabolic waste from the interstitial space of the brain parenchyma) as common linker between the brain and the periphery [1-2]. This highly polarized macroscopic system is primarily active during non rapid eye movement (non-REM) sleep and states of high slow-wave activity. It is inextricably linked to sleep, to the extent that flow appears to stop with the onset of wakefulness. The hypothesis of an imbalance of glymphatic system as linker between the brain and the periphery has been funded (PRIN 2020SCBBN2_005 for the research "Glymphatic system: a new player in the gut-brain axis. Natural resources to maintain homeostasis". Goal: To evaluate the relationship between gut dysbiosis and glymphatic system alteration and to characterize the healthy properties of a food supplement based on plant extracts for relieving gut disequilibrium and the associated disorders focusing on brain dysfunctions due to a deficient CSF diffusion and waste clearance (36 months). Role Co-PI).

Starting by this grant, Cristina Lanni has recently started up a new research, with the aim to better investigate the circadian-driven glymphatic system dynamics as an early driver of neurodegeneration. This working hypothesis has been recently awarded by the Alzheimer's Association (Grant AARG-23-1140660 for the research «Misalignment in circadian glymphatic system as trigger of neurodegeneration» Goal: To establish the threshold (or time window) beyond which the glymphatic system impairment and pathological waste deposition, due to circadian misalignment, becomes irreversible and triggers neurodegenerative processes (36 months). Role: PI). The characterization of the events and the relationship among gut inflammation, glymphatic influx alteration and brain waste product deposition will contribute in understanding how a peripheral inflammatory hit may disorganize and drive a process prodromal of neurodegeneration.

References

[1] Fagiani F, Di Marino D, Romagnoli A, Travelli C, Voltan D, Di Cesare Mannelli L, Racchi M, Govoni S, Lanni C. Signal Transduct Target Ther. 2022;7(1):41.
[2] Fagiani F, Baronchelli E, Pittaluga A, Pedrini E, Scacchi C, Govoni S, Lanni C. Front Mol Neurosci. 2022;15:937174.

Selected peer-reviewed publications

1. Fagiani F, Fulop T, Govoni S, **Lanni C**. The Fuzzy Border between the Functional and Dysfunctional Effects of Beta-Amyloid: A Synaptocentric View of Neuron-Glia Entanglement. Biomedicines. 2023 Feb 8;11(2):484. doi: 10.3390/biomedicines11020484.
2. Basagni F, Naldi M, Ginex T, Luque FJ, Fagiani F, **Lanni C**, Iurlo M, Marcaccio M, Minarini A, Bartolini M, Rosini M. Inhibition of β-Amyloid Aggregation in Alzheimer's Disease: The Key Role of (Pro)electrophilic Warheads. ACS Med Chem Lett. 2022 Oct 10;13(11):1812-1818. doi: 10.1021/acsmedchemlett.2c00410.
3. Fagiani F, Baronchelli E, Pittaluga A, Pedrini E, Scacchi C, Govoni S, **Lanni C**. The Circadian Molecular Machinery in CNS Cells: A Fine Tuner of Neuronal and Glial Activity With Space/Time Resolution. Front Mol Neurosci. 2022 Jul 1;15:937174. doi: 10.3389/fnmol.2022.937174.
4. Govoni S, Fagiani F, **Lanni C**, Allegri N. The Frailty Puzzle: Searching for Immortality or for Knowledge Survival? Front Cell Neurosci. 2022 Feb 17;16:838447. doi: 10.3389/fncel.2022.838447.
5. Fagiani F, Di Marino D, Romagnoli A, Travelli C, Voltan D, Mannelli LDC, Racchi M, Govoni S, **Lanni C**. Molecular regulations of circadian rhythm and implications for physiology and diseases. Signal Transduct Target Ther. 2022 Feb 8;7(1):41. doi: 10.1038/s41392-022-00899-y.
6. Fagiani F, Vlachou M, Di Marino D, Canobbio I, Romagnoli A, Racchi M, Govoni S, **Lanni C**. Pin1 as Molecular Switch in Vascular Endothelium: Notes on Its Putative Role in Age-Associated Vascular Diseases. Cells. 2021 Nov 24;10(12):3287. doi: 10.3390/cells10123287.

7. Fagiani F*, **Lanni C***, Racchi M, Govoni S. (Dys)regulation of Synaptic Activity and Neurotransmitter Release by β-Amyloid: A Look Beyond Alzheimer's Disease Pathogenesis. Front Mol Neurosci. 2021 Feb 24;14:635880. doi: 10.3389/fnmol.2021.635880. *both authors equally contributed
8. Fagiani F, Catanzaro M, Buoso E, Basagni F, Di Marino D, Raniolo S, Amadio M, Frost EH, Corsini E, Racchi M, Fulop T, Govoni S, Rosini M, **Lanni C**. Targeting Cytokine Release Through the Differential Modulation of Nrf2 and NF-κB Pathways by Electrophilic/Non-Electrophilic Compounds. Front Pharmacol. 2020 Aug 14;11:1256. doi: 10.3389/fphar.2020.01256.
9. Fagiani F*, **Lanni C***, Racchi M, Govoni S. Targeting dementias through cancer kinases inhibition. Alzheimers Dement (N Y). 6(1):e12044; 2020 doi: 10.1002/trc2.12044. *both authors equally contributed
10. Catanzaro M, Fagiani F, Racchi M, Corsini E, Govoni S, **Lanni C**. Immune response in COVID-19: addressing a pharmacological challenge by targeting pathways triggered by SARS-CoV-2. Signal Transduct Target Ther. 5(1):84; 2020. doi: 10.1038/s41392-020-0191-1.
11. **Lanni C**, Masi M, Racchi M, Govoni S. Cancer and Alzheimer's disease inverse relationship: an age-associated diverging derailment of shared pathways. Mol Psychiatry. 2020 doi: 10.1038/s41380-020-0760-2.
12. Serafini MM, Catanzaro M, Fagiani F, Simoni E, Caporaso R, Dacrema M, Romanoni I, Govoni S, Racchi M, Daglia M, Rosini M, **Lanni C**. Modulation of Keap1/Nrf2/ARE Signaling Pathway by Curcuma- and Garlic-Derived Hybrids. Front Pharmacol. 10:1597; 2020. doi: 10.3389/fphar.2019.01597.
13. Fagiani F*, **Lanni C***, Racchi M, Pascale A, Govoni S. Amyloid-β and Synaptic Vesicle Dynamics: A Cacophonic Orchestra. J Alzheimers Dis. 72(1):1-14; 2019. doi: 10.3233/JAD-190771. *both authors equally contributed
14. Basagni F, **Lanni C**, Minarini A, Rosini M. Lights and shadows of electrophile signaling: focus on the Nrf2-Keap1 pathway. Future Med Chem. 11(7):707-721; 2019. doi: 10.4155/fmc-2018-0423.
15. **Lanni C**, Fagiani F, Racchi M, Preda S, Pascale A, Grilli M, Allegri N, Govoni S. Beta-amyloid short- and long-term synaptic entanglement. Pharmacol Res. 139:243-260; 2019. doi: 10.1016/j.phrs.2018.11.018.
16. Catanzaro M, Corsini E, Rosini M, Racchi M, **Lanni C**. Immunomodulators Inspired by Nature: A Review on Curcumin and Echinacea. Molecules. 23(11):2778; 2018. doi: 10.3390/molecules23112778.
17. Bisceglia F, Natalello A, Serafini MM, Colombo R, Verga L, **Lanni C**, De Lorenzi E. An integrated strategy to correlate aggregation state, structure and toxicity of Aß 1-42 oligomers. Talanta. 188:17-26; 2018. doi: 10.1016/j.talanta.2018.05.062.
18. Serafini MM, Catanzaro M, Rosini M, Racchi M, **Lanni C**. Curcumin in Alzheimer's disease: Can we think to new strategies and perspectives for this molecule? Pharmacol Res. 124:146-155; 2017. doi: 10.1016/j.phrs.2017.08.004.
19. Simoni E, Serafini MM, Caporaso R, Marchetti C, Racchi M, Minarini A, Bartolini M, **Lanni C**, Rosini M. Targeting the Nrf2/Amyloid-Beta Liaison in Alzheimer's Disease: A Rational Approach. ACS Chem Neurosci. 8(7):1618-1627; 2017. doi: 10.1021/acschemneuro.7b00100.
20. Govoni S, Mura E, Preda S, Racchi M, **Lanni C**, Grilli M, Zappettini S, Salamone A, Olivero G, Pittaluga A, Marchi M. Dangerous Liaisons between Beta-Amyloid and Cholinergic Neurotransmission. Curr Pharm Des. 20(15):2525-38; 2014.
21. **Lanni C**, Necchi D, Pinto A, Buoso E, Buizza L, Memo M, Uberti D, Govoni S, Racchi M. Zyxin is a novel target for β-amyloid peptide: characterization of its role in Alzheimer's pathogenesis. J Neurochem. 125(5):790-9; 2013.
22. **Lanni C**, Racchi M, Memo M, Govoni S, Uberti D. p53 at the crossroads between cancer and neurodegeneration. Free Radic Biol Med. 52(9):1727-33; 2012.
23. Mura E, Zappettini S, Preda S, Biundo F, **Lanni C**, Grilli M, Cavallero A, Olivero G, Salamone A, Govoni S, Marchi M. Dual effect of beta-amyloid on α7 and α4β2 nicotinic receptors controlling the release of glutamate, aspartate and GABA in rat hippocampus. PLoS One. 7(1):e29661; 2012.
24. Stanga S, **Lanni C**, Govoni S, Uberti D, D'Orazi G, Racchi M. Unfolded p53 in the pathogenesis of Alzheimer's disease: is HIPK2 the link? Aging (Albany NY) 2(9):545-54; 2010.
25. Buoso E, **Lanni C**, Schettini G, Govoni S, Racchi M. beta-Amyloid precursor protein metabolism: focus on the functions and degradation of its intracellular domain. Pharmacol Res. 62(4):308-17; 2010.
26. **Lanni C**, Nardinocchi L, Puca R, Stanga S, Uberti D, Memo M, Govoni S, D'Orazi G, Racchi M. Homeodomain interacting protein kinase 2: a target for Alzheimer's beta amyloid leading to misfolded p53 and inappropriate cell survival. PLoS One. 2010 A;5(4):e10171.
27. Mura E, **Lanni C**, Preda S, Pistoia F, Sarà M, Racchi M, Schettini G, Marchi M, Govoni S. Beta-Amyloid: A Disease Target or a Synaptic Regulator Affecting Age-Related Neurotransmitter Changes? Curr Pharm Des.16(6):672-83; 2010.
28. Mura E, Preda S, Govoni S, **Lanni C**., Trabace L, Grilli M, Lagomarsino F, Pittaluga A, Marchi M. Specific Neuromodulatory Actions of Amyloid-beta on Dopamine Release in Rat Nucleus Accumbens and Caudate Putamen. J Alzheimers Dis. 19(3):1041-53; 2010.
29. Colombo R, Carotti A, Catto M, Racchi M, **Lanni C**, Verga L, Caccialanza G, De Lorenzi E. CE can identify small molecules that selectively target soluble oligomers of amyloid beta protein and display antifibrillogenic activity. Electrophoresis, 30(8):1418-29, 2009.

30. **Lanni C**, Racchi M, Mazzini G, Ranzenigo A, Polotti R, Sinforiani E, Olivari L, Barcikowska M, Styczynska M, Kuznicki J, Szybinska A, Govoni S, Memo M, Uberti D. Conformationally altered p53: a novel Alzheimer's disease marker? Mol Psychiatry, 13(6): 641-647; 2008.