Introduction

Prof. Olivotto chairman of the symposium and Prof. Marchionni, head of the Cardiothoracovascular Department - Careggi Hospital - Florence, opened the congress, by greeting the audience coming from many European countries and beyond. Prof. Marchionni highlighted the high scientific level of the congress thanks to the attendance of many of the worldwide top researches in the cardiological field, giving the chance for sharing information and knowledge on epidemiology, clinical and experimental studies, pathophysiology, biology, genetics about cardiomyopathies.

To follow the presentations of this congress, click on the link below:
“translational is the way to go, for a better understanding of the disease” Prof. Yacoub pointed out at the beginning of his lecture. The speaker, coming from London (UK), went deeper in his talk by highlighting the role played by translational research in crossing the so-called “valley of death”. In order to better explain this concept, the speaker briefly presented some researchers like Robert Koch, Sir John Gurdon, Fred Sanger and others, pointing to their discoveries in the field of Medicine that have completely changed the history of human being. Finally, Prof. Yacoub highlighted the relevance gained by translational research in the field of Cardiomyopathies. He spoke also about the role played by biomechanics and biotechnology and more in particular about CRISPER-CAS9. In conclusion, the speaker pointed out that Translational Research in Cardiomyopathy is accelerating at unprecedented rate and promises to deliver soon new discoveries able to improve the quality of life of these patients.

- What is translational research?
- Why translational research is relevant for Cardiomyopathy?
- What is the role of Biomechanics?
- What’s about the new tools like in vivo CRISPER-CAS9?

To follow the presentations of this congress, click on the link below:
Why do we need more genetic research in cardiomyopathy?

Prof. Ware from London (UK), spoke about cardiomyopathy and the need for more genetic research, by presenting very interesting data on the clinical impact of genetic testing in cardiomyopathies. More in particular the speaker talked about the predictive testing to be performed on relatives in order to identify the possible presence of mutations and about precision medicine able to tailoring the treatment, based on the characteristics of any patient. Prof. Ware presented many data on precision medicine and its application like genotype stratification and targeted therapies for patients affected by recessive and dominant diseases. Finally, the speaker talked about the in vivo effect of Myh6 R403Q silencing abnormal mRNA, that leads to the significant reduction of fibrosis in patients. Prof. Ware concluded his lecture, speaking about two topics: the gene editing in muscular dystrophy and MYK-461, that is a small molecule inhibitor of sarcomere contractility.

- What is the clinical impact of genetic testing in cardiomyopathies?
- What is precision medicine for patients affected by genetic diseases?
- What’s about the target therapies in recessive diseases?
- What are the main therapies discovered for patients affected by dominant genetic diseases?
- Why is not possible to interpret variants even in real cardiomyopathy genes?

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Insight from international registries

The international registries were the topics Prof. Girolami and Prof. Y. Ho spoke about in their talk. The speakers coming from Florence (I) and from Boston (USA), presented very interesting data on the genetic basis of HCM and the role played by genetics in helping precision medicine. More in particular Prof. Girolami presented very interesting data on families affected by cardiomyopathies, these data were performed by applying the NGS strategy. In the main part of her presentation, Prof. Girolami spoke about the sarcomeric human cardiomyopathy registry by presenting very interesting data on the variants’ revaluation and its impact on the clinical management of patients. Prof. Ho spoke about the role of genetics for improving the “precision” in medicine. The speaker presented very interesting data on the role played by genetics in improving diagnosis, individualizing treatments and preventing the onset of HCM.

- What is the main result of the NGS strategy application?
- What’s about the tsunami of variants from the speaker point of view?
- What are the main results of the sarcomeric, human cardiomyopathy registry presented by Prof. Girolami?
- What governs symptom burden and adverse events, in patients affected by HCM?
- What’s about the outcomes with MYH7 mutations?
- How might sarcomere protein mutations cause HCM?
- What’s about HCM ♥Net Multicenter Clinical Network?
- What is the effect of MYK-461 on Fibrosis and Disarray?
- Is it possible to silence HCM gene mutations?

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Deep phenotyping of gene+/pheno- individuals

Deep phenotyping of gene+/pheno- individuals was the topic at the core of the lecture discussed by Prof. Michels from Rotterdam (Netherland). “What is phenotype negative in HCM?” with this question the speaker addressed the audience and with the aim to find an acceptable answer, she went deeper in her lecture by presenting very interesting data on these topics: family screenings, prevalence of disease in G+ family members, clinical outcome of relatives, HCM mutation carriers. In the main part of her talk, Prof. Michels spoke about ECG, morphological, functional, metabolic and pro-fibrotic features in gene+/hypertrophic patients. In conclusion, the speaker pointed out that pre-LVH findings in g+ subjects give valuable insight for a better knowledge of HCM pathophysiology, despite the need for more information on the natural history of g+/LVH- subjects.

- What is the prevalence of HCM in G+ family members?
- What are the times of development of HCM in G+ family members?
- What is the clinical outcome of the relatives?
- What are the functional features in G+/LVH- subjects?
- What are the pro-fibrotic changes in G+/LVH- subjects?

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New therapeutic targets and biomarkers for cardiac fibrosis

Prof. Cook, co-chairman of the symposium, talked about the new therapeutic targets and biomarkers for cardiac fibrosis. The speaker started his talk by addressing the audience with this question: “how do titin variants cause cardiomyopathy”? TTNtv occur in up to 25% of severe and familial DCM cases but also have been seen in 2% of general population, the speaker highlighted. Starting from this point, Prof. Cook went deeper in his lecture, by presenting a huge amount of data about TTNtv mutations, their location, and the molecular mechanisms underlying the cardiomyopathy. In the last part of his presentation, Prof. Cook spoke about mTORC signalling activation and its effect on the heart, leading to the loss of function in myocardial contractility and the onset of heart failure.

- Proximal vs distal variants – does it matter?
- Do proximal and distal truncations affect TTN RNA and protein in a similar or dissimilar way?
- What are the molecular mechanisms underlying titin cardiomyopathy?
- Do all TTNtv have an effect on the heart that can be revealed using advanced imaging?
- Titin truncating variants that can cause DCM are seen in the general population in ~1:200 people, but who is really at risk?

To follow the presentations of this congress, click on the link below:  
Lessons from cellular electrophysiology

Cardiomyopathies and their correlation with cellular electrophysiology was the topic of Dr. Coppini’s presentation. The speaker, coming from Florence (I), at the beginning of his speech, talked about the anatomic arrhythmogenic substrate in HCM myocardium, by highlighting that the premature ventricular complexes have their origin in single cells. Dr. Coppini went deeper in his talk, by presenting very interesting data on some topics, like the correlation between the action potentials’ prolongation and the onset of arrhythmias in cardiomyocytes or the ions flux alterations like Ca\(^{2+}\) and Na\(^{+}\) current increasing, driving cardiomyocyte remodelling. Finally, the speaker talked about the effect of the increased activation of calmodulin kinase II on the cardiomyocyte metabolism and the effect of T-tubular loss and disorganization on the cellular functions. In conclusion Dr. Coppini pointed out that the increased late Na\(^{+}\) current, plays a major role in cellular arrhythmogenesis in patients affected by HCM and that the mechanical measurements of myofibrils isolated from HCM patients affected by MYH7 mutations are at the basis of the future electrophysiological research in the field of HCM.

- What is the anatomic arrhythmogenic substrate in HCM myocardium?
- What are the determinants of AP prolongation in HCM?
- What is the effect of the INaL inhibition performed by ranolazine on APs?
- What are the main consequences of increased INaL in cardiomyocytes?
- What’s about HCM mouse models with cTnT mutations?
- What are the effects of INaL inhibition on myocardial remodeling in HCM?
- What are the consequences of INaL inhibition in HCM cardiomyocytes?

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Rationale for metabolic treatment of hypertrophic cardiomyopathy

Prof. Van Der Velden talked about hypertrophic cardiomyopathy and the rationale for its metabolic treatment. The speaker, coming from Amsterdam (Netherlands), presented very interesting data on the underlying mechanisms driven by mutations and leading to the onset of disease. “Does a sarcomere mutation cause inefficient sarcomere contraction”? With this question Prof. Van Der Velden addressed the audience at the beginning of her lecture and, in order to find a satisfying answer, she went deeper in her lecture, by presenting a huge amount of in vitro and in vivo data, all of them demonstrating that a sarcomere mutation can lead to a loss in myocardial contractile efficiency. In the last part of her presentation, the speaker talked about a new treatment characterized by the administration of perhexiline, a metabolic modulator, to HCM patients, highlighting that the metabolic shift from fatty acid to glucose performed by perhexiline can produce an increase in PCr/ATP ratio.

- What does a mutation have to do to cause disease?
- Does a sarcomere mutation cause inefficient sarcomere contraction?
- How is it possible to initiate a metabolic therapy at an early stage in asymptomatic mutation carriers?
- What is the carrier of the early stage diastolic dysfunction?

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Clinical development of novel treatment options

The clinical development of novel treatment options was the main topic of Prof. Olivotto’s presentation. The speaker, co-chairman of the symposium, presented very interesting data on the pharmacological treatment of HCM patients. At the beginning of his lecture, Prof. Olivotto spoke about the natural evolution of the disease and the effects produced by the pharmacological treatment. He presented data on the prevalence and the annual event rate in HCM patients and pointed out that the new candidate pharmacological products are designed for a better correction of the underlying causal defects. In the main part of his presentation, the speaker talked about the therapeutic targets in cardiomyopathy like late sodium currents and the effect of ranolazine in the Restyle-HCM trial. In the last part of his talk, Prof. Olivotto spoke about the adaptive designs for clinical trials to be conducted in HCM patients and about the role played by registries at the core of the cycle of quality in all the clinical processes, from the clinical evidence to the outcome evaluation.

- What’s about the treatment of a lifelong disease?
- How is it possible to assess the efficacy?
- What are the therapeutic targets in cardiomyopathy?
- What are the novel perspective in the pharmacological treatment of hypertrophic cardiomyopathy?
- What are the main cardiovascular end-point in HCM?
- What is the role of clinical registries in the cycle of quality?

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Novel imaging and peripheral biomarkers in cardiomyopathy: what is the added value?

Novel imaging and peripheral biomarkers of cardiomyopathy were the topics at the core of Prof. Prasad’s presentation. The speaker coming from London (UK), started his talk by highlighting that the diagnosis of patients affected by HCM is one of the big challenges, characterized by the need for an early detection, the discovery of novel therapeutic targets, a better timing of therapy, a better risk stratification, a better selection for device therapy and finally also the need for fiscal restraint. Prof. Prasad went deeper in his lecture by presenting very interesting data on imaging for the diagnosis of cardiomyopathy. In the last part of his presentation, the speaker talked about biomarkers and why there are useful in HCM patients. More in particular he presented data on NT-pro BNP, Troponin, ST2 and galectin. Prof. Prasad pointed out that, in order to perform a better diagnosis of HCM, it is necessary to use biomarkers together with imaging techniques. Finally, the speaker spoke about future opportunities and more in particular about the multiparametric biomarker analysis.

- What are the main challenges in HCM diagnosis?
- What’s new in HCM diagnosis?
- What’s about Hybrid PET/MRI in HCM patients?
- Could C13 Imaging be an Alternative in HCM diagnosis?
- What are the main limitations of Imaging applied to the HCM diagnosis?
- Why are Blood-Based Biomarkers useful in HCM diagnosis?

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Role of CMR mapping in myocardial tissue characterization

Prof. Bogaert, talked about CMR mapping and its role in the characterization of the myocardial tissue. The speaker coming from Leuven (Belgium), presented very interesting data on this technique, the so called “CMR myocardial mapping”, by highlighting its role as a novel biomarker. He spoke also about its clinical application in patients affected by cardiomyopathies. More in particular Prof. Bogaert presented a huge amount of data on these topics: the diagnostic accuracy, the geno-phenotyping relationship, the disease’s detection in an early phase, the follow-up of the disease progression, the differentiation with phenocopies, the prognosis assessment and finally the evaluation of the effect of novel treatment strategies.

- What are the clinical applications of CMR myocardial mapping in Cardiomyopathies?
- What’s about the improved accuracy in diagnosis?
- What’s about CMR mapping in Anderson-Fabry disease?
- What’s about the detection of Early or Pre-Clinical Disease?
- What are the main Phenocopies of HCM?
- What’s about the efficacy of Novel Treatment Strategies?

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3D CMR and atlasing the heart

3D CMR as a new technique able to atlas the heart, was the topic discussed by Prof. O’Regan. The speaker, coming from London (UK), presented very interesting data on this new technique giving an impressive demonstration of its potentiality for a better identification of the cardiovascular phenotypes. More in particular Prof. O’Regan went deeper in explaining the model at the basis of 3D CMR by speaking about covariates, candidate genes, 3D GWAS and prediction’s power when applied in HCM patients. The speaker pointed out that 3D CMR allows to create high-fidelity cardiovascular phenotypes through the application of computational models, provides a stronger signal for candidate gene and GWAS, offers mechanistic insight into remodeling and allows personalised predictive models for outcomes.

- What’s about covariates?
- What’s about the statistical model for identifying the candidate genes?
- What’s about 3D GWAS?
- What is the model for prediction, presented by the speaker?

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Prof. Trayanova from Baltimore (MA-USA), spoke about the integration of morphology and function, in order to predict the arrhythmic risk. More in particular the speaker presented very interesting data on the personal virtual heart that is a program supported by an NIH director’s pioneer award, aiming to the creation of a patient-specific simulation environment for a better diagnosis and treatment of rhythm disorders of the heart in patients affected by structural myocardial diseases. The speaker went deeper in her presentation by explaining how, starting from a specific patient cardiac MRI, she is able to create the personal virtual heart of that patient. Finally, Prof. Trayanova spoke about a prospective study running in patients who undergo to ICD for primary prevention, by presenting very interesting data. In conclusion, the speaker pointed out that the personal virtual heart is a transformative approach, it brings computational modelling of individual heart dysfunction to the clinic as a guidance for therapies and it represents a paradigm shift in cardiac patient care.

- What’s about the personal virtual heart program?
- What are the main advantages in applying this new specific methodology?
- What’s about the model generation?
- What’s about the proof of concept study?
- What’s about the prospective study on patients undergoing to ICD?

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What can we learn from genotype-phenotype correlations in DCM?

The genotype-phenotype correlations in DCM was the topic discussed by Dr. Tayal. More in particular the speaker, coming from London (UK), presented very interesting data on the current and future clinical DCM treatment paradigm, by highlighting the unmet needs of DCM patients. In the main part of her presentation the speaker talked about genetics as the way for a better knowledge of the pathogenic mechanisms underlying DCM. In conclusion, the speaker pointed out that TTNtv can be useful for a better phenotypic characterization of DCM patients, it is independently predictive of early arrhythmia, and it is very useful for an early identification of high risk DCM patients.

- Could alcohol be a phenotypic modifier of TTNtv cardiomyopathy?
- What are the main DCM unmet needs?
- What is the main clinical problem linked with DCM?

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Characterization of copy number variants in dilated cardiomyopathy patients and healthy controls

Dr. Mazzarotto from Florence (I), spoke about the characterization of copy number variants in DCM patients compared with healthy controls, by presenting very interesting data derived from a study performed on a family affected by DCM. The speaker presented very interesting data on the genome sequencing performed on an Egyptian family characterized by the presence of three children affected by a very severe early-onset form of DCM, in order to understand if the di-genetic etiology of DCM could be caused by the simultaneous alteration of tAB1+TTN.

- How many variants have been discovered through the genome sequencing performed by the speaker?
- What are the main candidate of disease discovered by the speaker?
- What’s about copy number variants?

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Re-evaluating the genetic architecture of cardiomyopathy

Re-evaluating the genetic architecture of cardiomyopathy, was the topic discussed by Dr. Walsh in his presentation. The speaker coming from London (UK), started his talk by presenting data on cardiomyopathies and on the genes involved in this mendelian disease. The speaker highlighted that HCM gene panels should be limited to those genes for which exist an evidence for an association with the disease and he pointed out that can be used a case-control analysis in order to improve the interpretation of variants in valid genes.

- Can big datasets validate disease genes?
- There are genes with no case excess?
- What’s about HCM and its role for non-sarcomeric genes?

To follow the presentations of this congress, click on the link below:
Carto-guided endomyocardial biopsy in the evaluation of acquired and inherited arrhythmogenic myocardial disorders

Dr. Pieroni from Arezzo (I), presented very interesting data on this topic: “the role of the carto-guided endomyocardial biopsy in the evaluation of arrhythmogenic myocardial disorders”. The speaker went deeper in his presentation, talking about the clinical indications, the research studies and the risks and limitations of the endomyocardial biopsy. Dr. Pieroni, presented also data on a clinical study running on patients affected by arrhythmogenic right ventricular cardiomyopathy and submitted to CARTO-guided EMB. Finally, the speaker talked about the 3D-EAM guided EMB in patients affected by Brugada syndrome.

- What’s about the application of 3D-EAM in Cardiomyopathies?
- What’s about 3D-EAM guided EMB in Brugada syndrome?

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Protean effects of Na-channels blockers in HCM, from Disopyramide to Ranolazine and Eleclazine

The main topics at the core of Dr. Ferrantini’s presentation, were the protean effects of the Na-channels blockers in HCM. The speaker, coming from Florence (I), presented very interesting data on the effects of the Na-channels blockers in HCM patients. Dr. Ferrantini, more in particular spoke about the effect of dysopyramide, ranolazine and eleclazine. The speaker highlighted that ranolazine and eleclazine may reduce septal hypercontractility and LVOT gradients during exercise with no effects on LV contractility at rest.

- What’s about the effects of INaL blockers under β adrenergic stimulation?
- What’s about the effects of disopyramide 5µM (Dis) in hCM patients?
- What’s about GS-967 0.5 µM on action potentials and Intracellular Ca2+ transients?

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Cardio Classifier - automating clinical variant interpretation for inherited cardiac conditions

Dr. Whiffin presented very interesting data on the interpretation of cardio-classifier automating clinical variant for inherited cardiac conditions. The speaker coming from London (UK) spoke about this new tool able to integrate data from multiple sources for a better interpretation of variants for inherited cardiac conditions. The speaker highlighted that one demo is available at www.cardioclassifier.org.

- What’s about cardio classifier?
- What’s about Variant Interpretation from the speaker point of view?
- What are the type of data integrated by this new tool?
- What’s about the disease specific annotations?

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ICD burden and novel perspectives for low-energy defibrillation: insights from ontogenetic studies

The main topic at the core of Dr. Sacconi’s presentation, was the ICD classification and new perspective for low-energy defibrillation. The speaker, coming from Florence (I), presented very interesting data on this new technique, by highlighting the possibility to customize the optical stimulation able to induce the cardioversion in patients affected by fibrillation. The speaker pointed out that with this technology it is possible to reduce the energy needed for cardioversion.

- What’s about the optical stimulation?
- What’s about the optically induced cardioversion?
- What is the Cardioversion successful rate in the data presented by the speaker?

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Population based deep phenotyping uncovers the effects of TTNtv

Dr. de Marvao from London (UK) spoke about deep phenotyping and TTNtv. The speaker presented very interesting data on 3D CMR and on the atlas-based 3D-CMR phenotyping able to detect Titin-truncated variants. More in particular Dr. de Marvao spoke about TTNtv in healthy humans and in patients, by highlighting that these mutations are present and penetrant in about 1% of the population and that the computational models of the genotype-phenotype relationships can play and important role in risk stratification and precision medicine.

- What’s about TTNtv across the population?
- What’s about CMR phenotyping?
- What’s about TTNtv in healthy humans?

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A Novel Portable Smartphone-based electrocardiograph for remote cardiovascular screening: the D-Heart Project

The D-Heart project was the topic at the core of Dr. Maurizi’s presentation. The speaker coming from Florence (I), talked about the relationship between the portable smartphone-based electrocardiography and the remote cardiovascular screening, by highlighting the clinical reliability and high quality tracing of this technique. In the main part of his presentation the speaker talked about the clinical validation performed in Senegal and presented also some data produced by this program, by highlighting that the application of this new technique leads to a better accuracy despite the use of a more friendly device. In conclusion Dr. Maurizi pointed out that this technique is a very useful tool for the de-medicalization of the ECG acquisition for screening purposes.

- What’s about the rate of cardiovascular related death in medium and low income countries by 2020?
- What’s about the mobile technologies development in Africa from the speaker point of view?
- What are the main characteristics of the D-Heart Solution?
- What’s about the study design and the primary objective related to the clinical validation presented by the speaker?

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