LSHM-CT-2006-037593

Cardiogenics

Integrated Project (IP)

Publishable final activity report 2006 to 2011

Period covered: from 1st October 2006 to 30th March 2011

Date of preparation: July, 4th 2011

Start date of project: 1st October 2006 Duration: 54 months

Project coordinator name: Prof. Dr. Heribert Schunkert

Project coordinator organisation name: University of Lübeck

1. Publishable Executive Summary

1.1 Identity Card

<table>
<tr><td colspan="3">   </td></tr>
<tr><td colspan="2">Project Identification</td><td>Not confidential</td></tr>
<tr><td colspan="3">Title of the project: Cardiogenics</td></tr>
<tr><td colspan="3">Acronym of the project: Cardiogenics</td></tr>
<tr><td colspan="2">Type of contract:</td><td>Total project cost:
11.471.846,00 Euro</td></tr>
<tr><td>Contract number
LSHM-CT-2006-037593</td><td>Duration
54 months</td><td>EU contribution: 9.999.903 €o</td></tr>
<tr><td>Commencement date
01/10/2006</td><td colspan="2">Period covered by the report
01/10/2006 to 30/03/2011</td></tr>
<tr><td>Project Coordinator:
Prof. Heribert Schunkert

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World wide web address:
http://www.Cardiogenics.eu</td><td colspan="2">Partners involved:
University of Leicester
Institute National de la santé et de la recherche medicale
University of Cambridge
University of Regensburg
Genome Research Limited
Sanquin Blood Supply Foundation
Helmholtz Zentrum München
University of Mainz
University of Uppsala
University of Belfast
University of Leeds
Associazione per lo studio della trombosi in cardiologia
Euroimmun AG
Trium Analysis Online GmbH
Technical University of Munich
University Hospital Regensburg
Universitätsmedizin der Johannes-Gutenberg Universität Mainz
National Institute of Health and Welfare, Helsinki</td></tr>
<tr><td colspan="3">Objective :

The heart is the organ that works non-stop to keep the blood circulation going. Its own oxygen needs are supplied by blood vessels that form a crown at the "top" of the heart, hence the name coronary artery disease (CAD) for constrictions in these arteries. Most often, these constrictions are caused by atherosclerosis, a condition when plaques of cholesterol and cells obstruct the blood flow. In the European Community, CAD is the leading cause of disability and death (744.000 persons per year). Despite significant advances in the management of CAD and its maior complication, heart attack, the burden of disease continues to increase, particularly in the Eastern European countries. Worldwide, CAD is predicted to be the leading cause of morbidity and mortality by 2020. The Cardiogenics project aims at discovering genetic variations leading to coronary artery disease, thereby uncovering the underlying disease mechanisms and helping develop new treatments. However, without the support of numerous heart patients and blood donors, whose samples are used by researchers to perform tests, these goals would remain unattainable.</td></tr>
</table>

1.1.1 Participant List

Partic.Role*	Partic. no.	Participant name	Participant short name	Country
CO	1	University of Lübeck	UNIHL	DE
CR	2	University of Leicester	ULEI	UK
CR	3	Institute National de la santé et de la recherche medicale	INSERM	F
CR	4	University of Cambridge	UCAM	UK
CR	5	University of Regensburg	UNIREG	D
CR	6	Genome Research Limited	WTSI	UK
CR	7	Sanquin Blood Supply Foundation	SANQUIN	NL
CR	8	Helmholtz Zentrum München	GSF	DE
CR	9 (10)	University of Mainz	JOGU	DE
CR	10 (11)	University of Uppsala	UU	SE
CR	11 (12)	University of Belfast	QUB	IR
CR	12 (13)	University of Leeds	UNIVLEEDS	UK
CR	13 (14)	Associazone per lo studio della trmbosi in cardiologia	ASTC	IT
CR	14 (15)	Euroimmun AG	EUAG	DE
CR	15 (16)	Trium Analysis Online GmbH	TRIUM	DE
CR	16 (17)	Technical University of Munich	TUM	DE
CR	17 (18)	University Hospital Regensburg	UHREG	DE
CR	18 (19)	Universitätsmedizin der Johannes-Gutenberg Universität Mainz	UMC	DE
CR	19 (20)	National Institute of Health and Welfare	THL	FIN

*CO = Coordinator

CR = Contractor

() = No. in CPF

2. Project objectives and major achievements

2.1 Overview of general project objectives, relation to the state-of-the-art

Atherosclerosis is a complex disease, caused by multiple genetic and environmental factors. Likewise, a multifactorial etiology applies to many of the underlying cardiovascular risk factors including hypercholesterolemia, hypertension, diabetes mellitus, and smoking addiction. Thus, endogenous (genetic) and exogenous (nutrition, physical activity, therapy etc.) mechanisms may affect the manifestation of atherosclerotic lesions either directly or indirectly via modulation of traditional risk factors. On a cellular level atherosclerosis is also complex characterized by endothelial dysfunction, lipid and matrix accumulation, smooth muscle cell (SMC) proliferation and migration, calcification, inflammation, and, finally, thrombus formation. In this scenario, the potential involvement of genetically modulated mechanisms is indicated.

For long, evaluation of the family history served as a guide to approach a patient's genetic risk for coronary events. Beyond the information conferred by a positive family history, identification of the underlying gene defects is thought to improve risk prediction and the knowledge of pathogenetic mechanisms.

Consequently, over the past three decades, a great deal of research has focused on defining such genetic components of myocardial infarction, atherosclerosis and its risk factors. The hope for the future is that knowledge of the genes and gene variants will lead to improvements in the diagnosis and treatment of coronary disease. Indeed, emerging data suggest that some of these gene variants identified over the last years allow improve genetic risk prediction with sufficient reproducibility for the clinical setting. Initially this research focused on candidate genes that hypothetically might affect known traits involved in the atherosclerotic process including the renin-angiotensin system, lipoprotein metabolism, inflammation or coagulation. Many of these attempts failed replication in consecutive studies. Another difficulty in this research was that, unlike Mendelian traits, genetic studies on complex cardiovascular disorders are complicated by variable cosegregation between the risk allele and the disease. In fact, many genetic variants associated with the disorders were found to

be relatively common in the overall population and, thus, – albeit to a variable degree – prevalent in both healthy and affected individuals.

Subsequently, genome wide linkage analysis searched without a priori hypothesis for chromosomal regions shared in family members with myocardial infarction. While this approach allowed identification of several chromosomal regions harboring myocardial infarction genes, it proved to be difficult to precisely define these.

However, success came with genome wide association (GWA) studies that most recently identified multiple gene variants reproducibly associated with coronary heart disease, hypercholesterolemia, or diabetes mellitus. Surprisingly, most of the genes identified thus far were not expected to play a role in the development of atherosclerosis. Thus, an important task for the immediate future is to understand the fundamental pathophysiological mechanisms affected by these genes in the development of atherosclerosis. Accordingly, functional information on these genetic factors and related gene expression as well as protein expression patterns is very much in need. Subsequently, genetic research may enhance diagnostic testing and development of new treatment targets.

2.2 Summary of the results, identification of major problems

In the year 2007, just at the beginning of Cardiogenics, three genome-wide association studies successfully identified variants on chromosome 9p21.3 to be strongly associated with risk of coronary artery disease and myocardial infarction. Since then, similar studies have firmly associated about 32 chromosomal loci all of which increase the susceptibility to coronary disease (Figure 1, 3).

This “gold rush of genomic discovery” was preceded by identification of the full human DNA sequence, followed by the cataloguing of common single nucleotide polymorphisms (SNPs) at these bases by SNP consortium, and development of arrays that allow simultaneous typing of millions such SNPs at relatively low cost. Based on these assets, genome-wide association studies compare the allele frequency of SNPs across the entire genome, with dense coverage of all chromosomes, between cases and controls. For high resolution and an unbiased view on the entire genome thousands of individuals have to be studied in this fashion. In order to meet this goal the research community formed international consortia and shared information for subsequent meta-analyses. Such joint effort ultimately resulted in successful identification of multiple genetic variants that are associated with an increased susceptibility to coronary disease (so-called risk alleles).

Genome-wide arrays preferentially contain SNPs that are frequently found in a population as those offers the highest statistical power to detect association. Accordingly, almost all currently identified risk alleles for coronary disease are common (Figure 1). For example, in an individual of European descent the probability to carry 1 or 2 risk alleles at the chromosome 9p21.3 locus is 51 and 27%, respectively. Thus, only 22% of Europeans are free of this specific genetic risk factor for myocardial infarction. Given the large number and the high frequency of risk alleles that have been identified thus far, virtually every person in our population carries multiple genetic variants that increase susceptibility to coronary disease.

Each risk allele increases the probability of coronary disease only by a relatively small margin, i.e. 5-15 relative percent per allele. There are two exemptions. One is a rare allele on chromosome 6q25.3 that markedly increases lipoprotein (a) levels and coronary disease risk by 54% (Figure 1). The other one is the relatively frequent one on chromosome 9p21.3, which increases risk by 29% per allele (Figure 1).

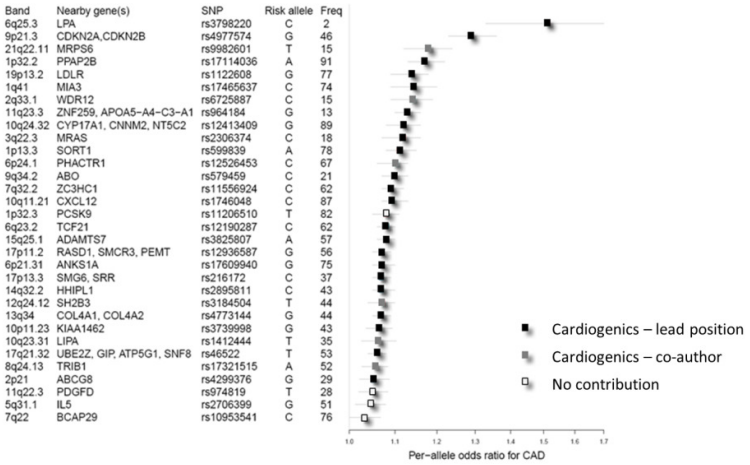

Figure 1. Shown are all known CAD/MI risk SNPs (status July 2011) and their respective effect sizes. 27 out of these 32 risk SNPs have been identified by contribution of Cardiogenics members either in lead position (22) or as co-author (5).

SNPs that are associated with a disease in a genome-wide association study are not necessarily causal. Rather, they mark a small region on a chromosome (a linkage disequilibrium block), in which such causal variant is likely to be located. Thus, a challenge that immediately follows the discovery of association between a SNP and a disease is the search for the affected gene and the causal variant(s) at the chromosomal locus. Studies that link SNPs with expression levels of genes (eQTL analysis) have been instrumental in this respect. The findings from these studies (conducted within Cardiogenics) suggest that at least some of the risk alleles affect expression of nearby genes in a quantitative and tissue-specific fashion and thereby modify the risk of coronary disease.

The rapidly growing list of chromosomal regions and genes associated with the risk of coronary disease is surprising in many aspects. Firstly, some of the strongest genetic effects were found in regions without known protein-coding genes. Secondly, only a minority of genetic variants showing clear association with coronary disease were also found to be associated with one of the traditional risk factors. Thirdly, in regions where annotation of the association signal to a specific gene was possible, only a few of these genes had previously been implied in the pathogenesis of coronary disease (ADAMTS7, COL4A1/A2, etc.). Finally, some of the loci did not only affect coronary disease risk but also multiple other, seemingly unrelated phenotypes (pleiotropy).

2.3 Work performed, results achieved so far

Completion of the human genome reference sequence in 2004, the systematic cataloguing of sequence variation by the SNP consortium and the HapMap project in 2005, as well as novel high throughput technologies for SNP typing now enable interrogation of the genome with ~80% coverage currently using 500,000 – 1,000,000 SNPs simultaneously. These technological advances opened a new era of the exploration of common diseases like coronary artery disease and myocardial infarction. Indeed, in the past years four independent GWA studies were completed on coronary artery disease (Helgadottir et al. 2007; McPherson et al. 2007; Samani et al. 2007). Most excitingly, all studies revealed uniformly that a single chromosomal locus (9p21.3) confers the strongest association with coronary artery disease and myocardial infarction. Six additional loci were identified by the Cardiogenics Consortium on chromosome 1 (1p13.3 and 1q41), chromosome 2 (2q36.3), chromosome 6 (6q25.1), chromosome 10 (10q11.21), and chromosome 15 (15q22.33) (Samani et al. 2007). To date for chromosome 9p21.3 large studies showing subsequent replication of the original association signal in different populations are published (Broadbent et al. 2007; Helgadottir et al. 2008). In addition, the Cardiogenics consortium has conducted a large-scale replication study for the above mentioned loci and the confirmed the 1p13.3, 1q41, 6q25.1, 9p21.3, and 10q11.21 in further 26,000 cases and controls (CAD consortium, under review).

Interestingly, the same chromosomal region on chromosome 9p21.3 but not the same SNPs are repeatedly associated with type 2 diabetes a risk factor for coronary artery disease. Moreover, the locus at chromosome 1p13.3 displayed strong association with LDL levels in several GWAs and subsequent replication studies.

Initially, the locus on chromosome 9p21.3 was reported to be associated with type 2 diabetes in three out of five genome wide association studies for type 2 diabetes. McPherson et al. (2007), Helgadottir et al. (2007), and the Cardiogenics Consortium (Samani et al. (2007)) were the first to report strong evidence for association between SNPs at the same chromosomal region and coronary artery disease/myocardial infarction. Helgadottir et al. (2008) provided further data showing that this locus not only affects coronary artery disease/myocardial infarction risk but also affects the risk of abdominal aortic aneurysm (AAA), intracranial aneurysm, peripheral arterial disease, and LAA/cardiogenic stroke in many populations

(Helgadottir et al. 2008). These findings may extend our knowledge about the role of sequence variants within the chromosome 9p21.3 region and show that it is not restricted to atherosclerotic diseases.

In parallel, Broadbent et al. (2007) reported that a large anti-sense non-coding RNA gene (ANRIL) collocates with the high-risk haplotype at chromosome 9p21.3 and that this gene is expressed in tissues and cell types that are affected by atherosclerosis and is therefore a prime candidate gene for the chromosome 9p21.3 coronary artery disease/myocardial infarction locus.

In the last five years a total of 60,000 patients with cardiovascular diseases and more than 70,000 controls are genotyped for SNPs at this locus making this gene region the most often replicated coronary artery disease/myocardial infarction region ever studied.

However, the above mentioned genetic variants explain only a small proportion of the inherited component (missing heritability) of CAD and MI.

One major reason is likely to lie in the complex nature of atherosclerosis where we expect that many genetic factors individually contribute only small effects to disease manifestation. In fact, for a typical genome-wide association study (GWAS) with about 1000 cases and controls, the power to detect any effects at stringent significance levels is poor. In order to overcome the constraint of relatively small sample sizes of individual studies, the Cardiogenics PIs formed a consortium to pool data across all published and multiple unpublished GWAS for CAD and MI. Of the various approaches to summarize evidence across studies we decided to conduct a type II meta-analysis on aggregated data from all studies.

While being organizationally and logistically challenging, screening for novel loci genome-wide utilizing meta-analysis provides substantially greater power. Specifically, our consortium under the header of Coronary ARtery DIsease Genome-wide Replication And Meta-analysis (CARDIoGRAM) comprises about ten times the number of cases and controls as compared to the largest published single GWAS. Through this, the power to detect small effects is substantially increased. For instance, even for genome-wide significance, the analysis will have power of about 80% for an odds ratio of only 1.1, provided that the minor allele comes with a frequency of at least 15%.

CARDIoGRAM is based on a core of previously successful collaborations between single participating studies. In this effort, genetic epidemiological methods across the participating samples were standardized, and a platform allowing summarized analyses on CAD, MI, and related phenotypes was created. It was anticipated that larger studies with almost 100.000 cases and controls will boost the identification of genetic risk loci.

During 2010 we worked on the joint database for the meta-analysis within the CARDIoGRAM consortium. The data was housed at SME TRIUM (Munich), safe data up- and download was secured. Intensive quality control of the uploaded data was undertaken before the meta-analysis was performed. Figure 1 shows the work flow of the analysis.

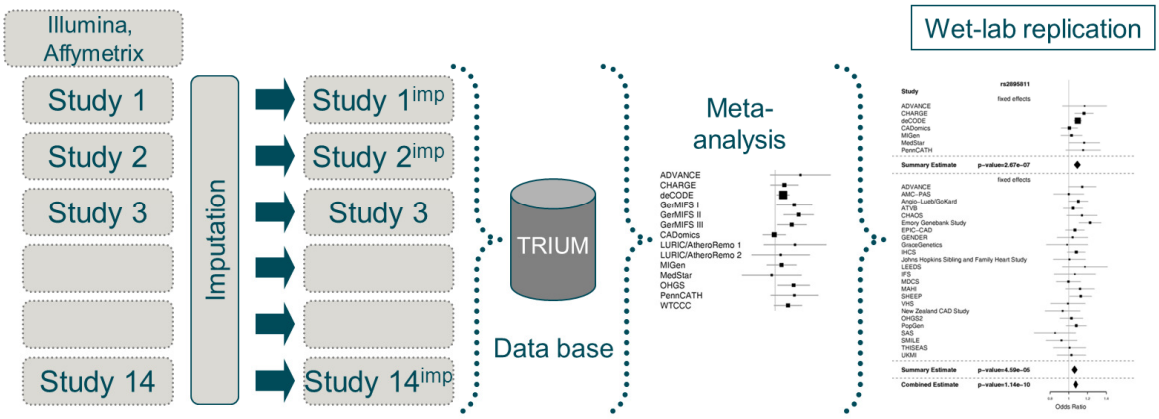

Figure 2. Schematic depiction of the work-flow of the CARDIoGRAM meta-analysis.

The meta-analysis identified a total of 23 SNPs ($p<10^{-6}$) which we followed up in a wet-lab replication step. Taking the number of loci into consideration, our replication study had >90% power to detect effect sizes observed in the GWAS meta-analysis. Of the 23 loci, 13 replicated using our *a priori* definition of a validated locus, that is, showing independent replication after Bonferroni correction and also achieving $P < 5 \times 10^{-8}$ in the combined discovery and replication data. The 13 new loci had risk allele frequencies ranging from 0.13 to 0.91 and were associated with a 6% to 17% increase in the risk of CAD per allele (Table 1).

Band	SNP	Gene(s) in region	Risk allele frequency (risk allele)	Meta-analysis		Replication		Combined analysis	
				P	*n*	*P*	*n*	OR (95% CI)	*P*
1p32.2	rs17114036	*PPAP2B*	0.91 (A)	1.43×10^{-8}	80,870	3.18×10^{-12}	52,356	1.17 (1.13–1.22)	3.81×10^{-19}
6p21.31	rs17609940	*ANKS1A*	0.75 (G)	2.21×10^{-6}	83,997	1.18×10^{-3}	53,415	1.07 (1.05–1.10)	1.36×10^{-8}
6q23.2	rs12190287	*TCF21*	0.62 (C)	4.64×10^{-11}	78,290	3.25×10^{-4}	52,598	1.08 (1.06–1.10)	1.07×10^{-12}
7q32.2	rs11556924	*ZC3HC1*	0.62 (C)	2.22×10^{-9}	80,011	7.37×10^{-10}	54,189	1.09 (1.07–1.12)	9.18×10^{-18}
9q34.2	rs579459	*ABO*	0.21 (C)	1.16×10^{-7}	77,138	7.02×10^{-8}	46,840	1.10 (1.07–1.13)	4.08×10^{-14}
10q24.32	rs12413409	*CYP17A1, CNNM2, NT5C2*	0.89 (G)	1.47×10^{-6}	80,940	1.38×10^{-4}	48,801	1.12 (1.08–1.16)	1.03×10^{-9}
11q23.3	rs964184	*ZNF259, APOA5-A4-C3-A1*	0.13 (G)	8.02×10^{-10}	82,562	2.20×10^{-9}	52,930	1.13 (1.10–1.16)	1.02×10^{-17}
13q34	rs4773144	*COL4A1, COL4A2*	0.44 (G)	4.15×10^{-7}	77,113	1.31×10^{-3}	37,618	1.07 (1.05–1.09)	3.84×10^{-9}
14q32.2	rs2895811	*HHIPL1*	0.43 (C)	2.67×10^{-7}	63,184	4.59×10^{-5}	51,054	1.07 (1.05–1.10)	1.14×10^{-10}
15q25.1	rs3825807	*ADAMTS7*	0.57 (A)	9.63×10^{-6}	80,849	1.39×10^{-8}	48,803	1.08 (1.06–1.10)	1.07×10^{-12}
17p13.3	rs216172	*SMG6, SRR*	0.37 (C)	6.22×10^{-7}	57,235	2.11×10^{-4}	54,303	1.07 (1.05–1.09)	1.15×10^{-9}
17p11.2	rs12936587	*RASD1, SMCR3, PEMT*	0.56 (G)	4.89×10^{-7}	76,952	1.35×10^{-4}	52,648	1.07 (1.05–1.09)	4.45×10^{-10}
17q21.32	rs46522	*UBE2Z, GIP, ATP5G1, SNF8*	0.53 (T)	3.57×10^{-6}	83,867	8.88×10^{-4}	53,766	1.06 (1.04–1.08)	1.81×10^{-8}

Table 2: New loci for coronary disease (Schunkert et al. Nature Genetics 2011)

This large number of risk variants were only be identified through major efforts started and coordinated by the PIs of Cardiogenics. Figure 3 gives an overview of all CAD/MI risk genes identified since 2007 and their localization on the human chromosomes.

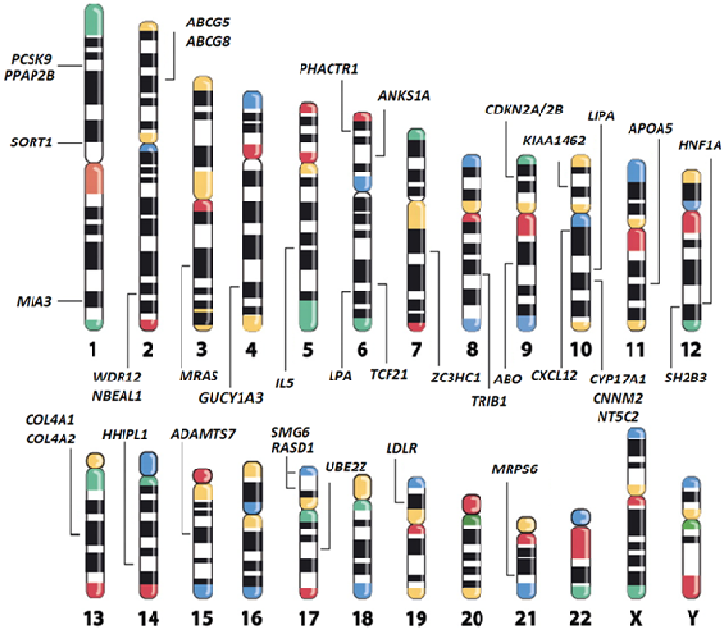

Figure 3. Schematic depiction of the human chromosomes and the localization of all known CAD/MI risk genes (status May 2011).

The genomic analysis of more than 135,000 individuals done with funding of the EU revealed three major findings.

- we identified 25 loci with firm association to CAD
- we found that only a minority of the established and new loci appear to act through traditional risk factors but that the majority reside in gene regions that were not previously suspected in the pathogenesis of CAD
- a substantial proportion of the CAD risk variants were also strongly associated with various other human disease traits in GWAS.

As a follow-up study of our genome wide association studies we conducted the first large-scale Population-based study to demonstrate the potential clinical utility of a genetic risk score for predicting incident CHD. Using prospectively followed cohorts from across Europe, we could demonstrate that genetic risk scores could refine risk classification when used in addition to the Framingham risk score. Although our study is the first to demonstrate the clinical utility of a genetic risk score in the general population, several limitations need to be considered. The list of genetic variants associated with CHD is steadily increasing such that our scores are likely incomplete. Despite this limitations, for the subset of men at intermediate risk, our scores significantly refined disease prediction. Our findings are a starting point for future meta-analyses with other cohorts to refine the predictive value of such genetic scores.

2.4 Expected end results

Identification and functional characterization of variants related to the risk of Coronary Artery Disease or Myocardial Infarction.

2.5 Intention for use and impact

Our findings facilitated a universal goal in modern healthcare, i.e. a better focus of early treatment on subjects with highly inherited risk for Coronary Artery Disease/ Myocardial Infarction. For this first) we gave insights into novel pathophysiologic pathways to be further followed-up in therapeutical studies, using a broad spectrum of methods (genomics, transcriptomics, proteomics, and metabolomics) and second) we developed diagnostic tests.

3.0 Dissemination of knowledge

3.1 Overview table 2006 to 2007

Planned/actual Dates	Type	Type of audience	Countries addressed	Size of audience	Partner responsible/involved
1/2007	Newsletter	Research	Europe	50	Coordinating office
20th December 2006	Management Meeting	Research	Europe	10	#1 - #6
20th -21 th February	Kick-off meeting	Research	Europe	50	All
03/2007	Cardiogenics Flyer	General Public	Worldwide	Unknown	Coordinating office
04/2007	Exhibition (Annual Meeting deutsche Gesellschaft für Kardiologie)	Higher education	Germany	500	Coordinating office
01/2007	Project Web-Site	General Public	Worldwide	Unknown	Coordinating office and # 15
18.07.2007	Press release, News (Tagesschau)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
18.07.2007	Press release Informationsdienst Wissenschaft Meilenstein in der Herzinfarkt-Forschung	Higher education	Worldwide	Unknown	Coordinating office
18.07.2007	Press release (Lübecker Nachrichten) Forscher finden sieben Risiko-Gene für Herzinfarkt	General Public	Germany	250000 (printed Press in Lübeck, Germany)	Coordinating office
18.07.2007	Press release (The new England journal of medicine) Genomewide Association Analysis of Coronary Artery Disease	Higher education	Europe	Unknown	Coordinating office
19.07.2007	Press release (Süddeutsche Zeitung) Erbanlagen können das Herzinfarkt-Risiko dramatisch erhöhen	General Public	Germany	unknown (printed Press in Germany)	Coordinating office

18.07.2007	Press release (Tagesspiegel) Veränderte Gene erhöhen das Herzinfarktrisiko		Germany	unknown (printed Press in Germany)	Coordinating office
12/2007	Media Briefing News American Heart Association	Higher education	Worldwide	Unknown	#1- # 6
2007	Publication Genomewide association analysis of coronary artery disease see Annex 2.1	Higher education	Worldwide	unknown	#1- # 6
2007	Publication (Repeated Replication and a Prospective Meta-Analysis of the Association Between Chromosome 9p21.3 and Coronary Artery Disease) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2007	Publication (Drinking from the fire hose--statistical issues in genomewide association studies.) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2007	Publication (Scanning the genome for coronary risk) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2007	Publication (Familial aggregation of left main coronary artery disease and future risk of coronary events in asymptomatic siblings of affected patients) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2007	Press (The Wellcome Trust Case Control and Cardiogenics Consortiums Identify Key Genes in Coronary Artery Disease with Affymetrix Technology) see Annex 2.1	General Public	Worldwide	Unknown	Coordinating office

2007	Publication (Lack of association between the MEF2A gene and myocardial infarction.) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2007	Publication (The lipoprotein subfraction profile: heritability and identification of quantitative trait loci) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
2008	Publication (An alternative splice variant in ABCC6, the gene causing dystrophic calcification, leads to protein deficiency in C3H/He mice) see Annex 2.1	Higher education	Worldwide	Unknown	#1- # 6
1/2008	Newsletter	Research	Europe	50	Coordinating office
15 -18 February 2008	Fourth EU–US Symposia Series, AAAS 2008	Research	Worldwide	5000	Coordinating office

3.2 Overview table 2007 to 2008

Planned/actual Dates	Type	Type of audience	Countries addressed	Size of audience	Partner responsible/involved
10/2007	Project Meeting, Leicester	Research	Europe	10	#1 - #6
12/2007	Media Briefing News American Heart Association (http://www.americanheart.org/presenter.jhtml?identifier=3052697)	Higher education	Worldwide	Unknown	#1- # 6
01/2008	Newsletter	Research	Europe	50	Coordinating office
01/2008	Press Release Informationsdienst Wissenschaft „Cardiogenics: Forschungsergebnisse zur Vererbung des Herzinfarktes weltweit führend - Wissenschaftlergruppe unter Lübecker Leitung auf der Bestenliste der American Heart Association“ (http://idw-online.de/pages/de/news241799)	Higher education	Worldwide	Unknown	Coordinating office

01/2008	Press release, Apothekenumschau „Kein Gentest für alle“ (http://www.presseportal.de/pm/52678/1126012/wort_und_bild_apotheken_umschau/rss)	General Public	Germany	Unknown	Coordinating office
02/2008	Fourth EU–US Symposia Series, AAAS 2008	Research	Worldwide	5000	Coordinating office
03/2007	Exhibition (Annual Meeting deutsche Gesellschaft für Kardiologie)	Higher education	Germany	500	Coordinating office
03/2008	Press Release, interview GesundheitPro „Kein Gentest für alle“ (http://www.gesundheitpro.de/A080303GOK0R067314)	General Public	Germany	Unknown	Coordinating office
03/2008	Press Release, interview NDR TV “Gefährliche Enge in den Adern – Arteriosklerose“ (http://www3.ndr.de/ndrtv_pages_std/0,3147,OID4211508,00.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
04/2008	Press Release, interview NDR TV “In der Suppe, in der Luft, in der Wanne - Wie viel Salz ist gesund?“ (http://www3.ndr.de/ndrtv_pages_std/0,3147,OID4211508,00.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
04/2008	Newsletter	Research	Europe	50	Coordinating office
05/2008	Second Annual Meeting, Lübeck	Research	Europe	50	#1 - #16
07/2008	Management Meeting, Stansted	Research	Europe	10	#1 - #6
08/2008	Newsletter	Research	Europe	50	Coordinating office
09/2008	Press Release, Deutsches Ärzteblatt “Cholesterinlüge entkräftet” (http://www.aerzteblatt.de/pdf/105/36/a1812.pdf)	Higher education	Germany	Unknown	Coordinating office
11/2008	Press Release, interview NDR TV “Stummer Infarkt: Wenn das Herz leise leidet“ (http://www3.ndr.de/ndrtv_pages_std/0,3147,OID4211508,00.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
2007	Publication (Familial aggregation of left main coronary artery disease and future risk of coronary events in asymptomatic siblings of affected patients) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5
2007	Publication (Head-to-head comparison of BNP and IL-6 as markers of clinical and experimental heart failure: Superiority of BNP) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5

2007	Publication (Polymorphisms in 33 inflammatory genes and risk of myocardial infarction – a system genetics approach) see Annex 2.1	Higher education	Worldwide	unknown	#3 + #11
2008	Publication (Genetic variants within the LPIN1 gene, encoding lipin, are influencing phenotypes of the metabolic syndrome in humans) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5
2008	Publication (Lack of association between the MEF2A gene and myocardial infarction) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5 + #8
2008	Publication (Genome-wide association study identifies genes for biomarkers of cardiovascular disease: serum urate and dyslipidemia) see Annex 2.1	Higher education	Worldwide	unknown	2# + #6
2008	Publication (An alternative splice variant in Abcc6, the gene causing dystrophic calcification, leads to protein deficiency in C3H/He mice) see Annex 2.1	Higher education	Worldwide	unknown	#1
2008	Publication (The lipoprotein subfraction profile: heritability and identification of quantitative trait loci) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5
2008	Publication (Congestive heart failure is a common disease with complex inheritance – new perspectives through genome wide association studies) see Annex 2.1	Higher education	Worldwide	unknown	#1
2008	Publication (Characterization of the GNAQ promoter and association of increased Gq expression with cardiac hypertrophy in humans) see Annex 2.1	Higher education	Worldwide	unknown	#1
2008	Publication (Repeated replication and a prospective meta-analysis of the association between chromosome 9p21.3 and coronary artery disease) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2008	Publication (Association of common polymorphisms in GLUT9 gene with gout but not with coronary artery disease in a large case-control study)	Higher education	Worldwide	unknown	#1 + #5

	see Annex 2.1				
2008	Publication (Telomere length is shorter in healthy offspring of subjects with coronary artery disease: support for the telomere hypothesis) see Annex 2.1	Higher education	Worldwide	unknown	#2
2008	Publication (Genetic variation in members of the leukotriene biosynthesis pathway confer an increased risk of ischemic stroke: a replication study in two independent populations) see Annex 2.1	Higher education	Worldwide	unknown	#8
2008	Publication (Genetic variation in soluble epoxide hydrolase (EPHX2) is associated with an increased risk of ischemic stroke in white Europeans) see Annex 2.1	Higher education	Worldwide	unknown	#8
2008	Publication (Genome-wide association analysis identifies 20 loci that influence adult height) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #4 + #6 + #12
2008	Publication (Common variants near MC4R are associated with fat mass, weight and risk of obesity) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #4 + #6 + #8 + #12
2008	Publication (Cholesterol gene polymorphisms and cardiovascular events) see Annex 2.1	Higher education	Worldwide	unknown	#3
2008	Publication (Lifelong reduction of LDL-cholesterol related to a common variant in the LDL-receptor gene decreases the risk of coronary artery disease - a Mendelian Randomisation study) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2008	Publication (Coronary artery disease-associated locus on chromosome 9p21 and early markers of atherosclerosis) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #12
2008	Publication (Molecular signatures of cardiovascular disease risk: potential for test development and clinical application) see Annex 2.1	Higher education	Worldwide	unknown	#1

2008	Publication (Lack of association of genetic variants in the LRP8 gene with familial and sporadic myocardial infarction) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #3 + #5 + #9 + #11
2008	Publication (Elevated C-reactive protein in atherosclerosis - chicken or egg?) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2
2008	Publication (The impact of newly identified loci on coronary heart disease, stroke and total mortality in the MORGAM prospective cohorts) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #3 + #11
2008	Publication (The novel genetic variant predisposing to coronary artery disease in the region of the PSRC1 and CELSR2 genes on chromosome 1 associates with serum cholesterol) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #5
2008	Publication (Genetic variation in the arachidonate 5-lipoxygenase-activating protein (ALOX5AP) is associated with myocardial infarction in the German population) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5
2008	Publication (Novel susceptibility locus for coronary artery disease on chromosome 3q22.3) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2008	Publication (A genome-wide haplotype association study identifies the *SLC22A3/LPAL2/LPA* gene cluster as a strong susceptibility locus for coronary artery disease) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2008	Publication (A genome-wide association study identifies three loci associated with mean platelet volume) see Annex 2.1	Higher education	Worldwide	unknown	#4 + #6 + #8

3.3 Overview table 2008 to 2009

Actual Dates	Type	Type of audience	Countries addressed	Size of audience	Partner responsible/ involved
11/2008	Management Team - Meeting, Stansted	Research	Europe	10	#1 - #6
11/2008	Press Release, interview NDR TV “Stummer Infarkt: Wenn das Herz leise leidet“ (http://www3.ndr.de/ndrtv_pages_std/0,3147,OID4211508,00.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
12/2008	Newsletter	Research	Europe	50	Coordinating office
01/2009	Newsletter	Research	Europe	50	Coordinating office
02/2009	EU–US Symposia Series, AAAS 2009	Research	Worldwide	5000	Coordinating office
02/2009	Press Release University of Luebeck “Hattrick bei der Entschlüsselung von Herzinfarktgenen“ http://www.mu-luebeck.de/aktuelles/pdfldateien/090205ge.pdf	Higher education	Worldwide	unknown	Coordinating office
02/2009	Press Release Deutsches Ärzteblatt „Neue Gene erklären den Herzinfarkt“ http://www.aerzteblatt.de/v4/news/news.asp?id=35362"]aerzteblatt.de	Higher education	Worldwide	unknown	Coordinating office
02/2009	Press Release Informationsdienst Wissenschaft „Neue Ansätze zur Vererbung von Herzinfarkt gefunden“ http://idw-online.de/pages/de/news300301	Higher education	Worldwide	unknown	#1 - #6
02/2009	Press-Links: “*Hattrick in deciphering of heart attack genes”* http://gesundheit-adhoc.de/index.php?m=1&showPage=1&id=5874 http://www.analytik.de/meldungen/2483.html http://www.curado.de/Herzinfarkt/Hattrick-bei-der-Entschluesselung-von -Herzinfarktgenen-10758/ http://dradio.de/dlf/sendungen/forschak/917112/"]dradio.de http://www.hl-live.de/aktuell/textstart.php?id=50367 http://www.schleswig-holstein.de/Wissenschaft/DE/Service/Aktuelles/090208UniLuebeckForschungHerzinfarkt.html http://www.shz.de/schleswig-holstein/artikeldetail/article/111/kein-freibrief-fuer-ungesunden-lebenswandel.html	Higher education	Worldwide	unknown	#1 - #6

	http://www.taz.de/1/archiv/print-archiv/printressorts/digi-artikel/?ressort=na&dig=2009%2F02%2F11%2Fa0176&cHash=23fe670534 http://wissenschaft-online.de/artikel/981356 http://www.focus.de/gesundheit/ticker/herz-kreislauf-entschluesselung-weiterer-herzinfarktgene-liefert-neue-therapieansaetze_aid_369670.html http://www.kn-online.de/schleswig_holstein/aus_dem_land/74722_Forscher entdeckten_Herzinfarktgene.html http://www.schattenblick.de/infopool/medizin/fakten/mz2n3178.html http://www.diabsite.de/aktuelles/nachrichten/2009/090208.html http://www.monstersandcritics.de/artikel/200907/article_124023.php/Entschl%C3%BCsselung-weiterer-Herzinfarktgene-liefert-neue-Therapieans%C3%A4tze http://www.ratschlag24.com/index.php/entschlsselung-weiterer-herzinfarktgene-liefert-neue-therapieanstze-_84748/ http://www.net-tribune.de/article/130209-83.php http://www.pressrelations.de/new/standard/result_main.cfm?pfach=1&n firmanr_=115875&sektor=pm&detail=1&r=355760&sid=&aktion=jour pm&quelle=0 http://health.usnews.com/articles/health/healthday/2009/02/08/genetic-test-for-heart-disease-risk-in-the-works.html http://news.health.com/2009/02/09/genetic-test-heart-disease-risk-works/ http://www.koamtv.com/Global/story.asp?S=9809215 http://www.kttc.com/Global/story.asp?S=9809215&nav=menu134826 http://www.lex18.com/Global/story.asp?S=9809215&nav=EQls http://www.empowher.com/news/2009/02/08/genetic-test-heart-disease-risk-works http://www.theheart.org/article/939879.do http://www.bild.de/BILD/ratgeber/gesund-fit/2009/02/12/neue-herzinfarkt-gene/entdeckt-neue-therapieformen-bessere-vorbeugung http://www.sueddeutsche.de/054389/180/2754605/Tief-ins-Erbgut-geblickt.html http://www.krankenkassenratgeber.de/news/gesundheit/neue-ansaetze-zur-vererbung-von-herzinfarkt-gefunden.html				
03/2009	Press Release, interview NDR TV „Essen als Medizin: Speisen für ein gesundes Herz“ http://www3.ndr.de/sendungen/visite/archiv/ernaehrung/essen136.html	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
04/2009	Exhibition (Annual Meeting deutsche Gesellschaft für Kardiologie)	Higher education	Germany	500	Coordinating office
04/2008	Third Annual Meeting, Lübeck	Research	Europe	50	#1 - #16

08/2009	Press Release HL-live „Lübecker Professor erforscht Herzinfarkt-Risiko“ (http://www.hl-live.de/aktuell/text.php?id=25925)	Higher education	Germnay	unknown	Coordinating office
09/2009	European Society of Cardiology Congress, Barcelona	Research	Europe	25.000	Coordinating office
2008	Publication (Elevated C-reactive protein in atherosclerosis--chicken or egg?) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2
2008	Publication (The novel genetic variant predisposing to coronary artery disease in the region of the PSRC1 and CELSR2 genes on chromosome 1 associates with serum cholesterol) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #5
2009	Publication (Genetic Investigation of ANthropometric Traits Consortium. Six new loci associated with body mass index highlight a neuronal influence on body weight regulation) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #4 + #6 + #8
2009	Publication (A genome-wide association study identifies three loci associated with mean platelet volume) see Annex 2.1	Higher education	Worldwide	unknown	#4 + #6 + #8
2009	Publication (Investigation of Crohn's disease risk loci in ulcerative colitis further defines their molecular relationship. Gastroenterology) see Annex 2.1	Higher education	Worldwide	unknown	#4 + #6
2009	Publication (New susceptibility locus for coronary artery disease on chromosome 3q22.3) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2009	Publication (Genome-wide haplotype association study identifies the SLC22A3-LPAL2-LPA gene cluster as a risk locus for coronary artery disease) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2009	Publication (Genome-wide association of early-onset myocardial infarction with single nucleotide polymorphisms and copy number variants) see Annex 2.1	Higher education	Worldwide	unknown	#1 - #16
2009	Publication	Higher	Worldwide	unknown	#1 + #2 + #3 + #12

	(The impact of newly identified loci on coronary heart disease, stroke and total mortality in the MORGAM prospective cohorts) see Annex 2.1	education			
2009	Publication (A novel variant on chromosome 7q22.3 associated with mean platelet volume, counts, and function) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #4 + #6 + #8
2009	Publication (Large scale association analysis of novel genetic loci for coronary artery disease) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #3 + #5 + #6
2009	Publication (Functional genomics in zebrafish permits rapid characterization of novel platelet membrane proteins) see Annex 2.1	Higher education	Worldwide	unknown	#4 + #7
2009	Publication (Genome-wide association study identifies eight loci associated with blood pressure) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #6 + #7 + #8
2009	Publication (Genome-wide association scan meta-analysis identifies three Loci influencing adiposity and fat distribution) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #6 + #8
2009	Publication (Meta-analysis of 28,141 individuals identifies common variants within five new loci that influence uric acid concentrations) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #8
2009	Publication (Regional differences in the prevalence of left ventricular hypertrophy within Germany) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #8
2009	Publication (Lack of association between a common polymorphism near the INSIG2 gene and BMI, myocardial infarction, and cardiovascular risk factors) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5
2009	Publication (The discovery of genes implicated in myocardial infarction) see Annex 2.1	Higher education	Worldwide	unknown	# 4
2009	Publication	Higher	Worldwide	unknown	#1 + #2 + #13

	(Genetic Loci associated with C-reactive protein levels and risk of coronary heart disease) see Annex 2.1	education			
2009	Publication (Genetic variation at chromosome 1p13.3 affects sortilin mRNA expression, cellular LDL-uptake and serum LDL levels which translates to the risk of coronary artery disease) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #2 + #5 + #8
2009	Publication (Genetic variants associated with cardiac structure and function: a meta-analysis and replication of genome-wide association data) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #8 + #10
2009	Publication (Common genetic variation near the phospholamban gene is associated with cardiac repolarisation: meta-analysis of three genome-wide association studies) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #6
2009	Publication (No association of two functional polymorphisms in human ALOX15 with myocardial infarction) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #5 + #8
2009	Publication (A genome-wide association study identifies a novel locus on chromosome 18q12.2 influencing white cell telomere length) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #6
2009	Publication (Identifying needs and opportunities for advancing translational research in cardiovascular disease) see Annex 2.1	Higher education	Worldwide	unknown	#1
2009	Publication (A functional genomics approach reveals novel quantitative trait loci associated with platelet signaling pathways) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #4 + #6
2009	Publication (Association between degenerative aortic valve disease and long-term exposure to cardiovascular risk factors: results of the longitudinal population-based KORA/MONICA survey) see Annex 2.1	Higher education	Worldwide	unknown	#1 + #8
2009	Publication	Higher	Worldwide	unknown	#1 + #5

	(Genetic determinants of circulating sphingolipid concentrations in European populations) see Annex 2.1	education			
2009	Publication (Concept, design and implementation of a cardiovascular gene-centric 50 k SNP array for large-scale genomic association studies) see Annex 2.1	Higher education	Worldwide	unknown	#2 + #4 + #13

3.2 Overview table 2009 to 2011

Actual Dates	Type	Type of audience	Countries addressed	Size of audience	Partner responsible/ involved
10/2009	Management Team - Meeting, Stansted	Research	Europe	10	#1 - #6
12/2009	Management Team - Meeting, Stansted	Research	Europe	10	#1 - #6
12/2009	Newsletter	Research	Europe	50	CO
01/2010	Press Release, interview; NDR TV “Frauenherzen schlagen anders“ (http://www.ndr.de/fernsehen/sendungen/visite/media/visite4274.html)	General Public	Germany	Unknown (TV in Germany, Austria)	CO
01/2010	Press Release, interview; NDR TV “Abenteuer Diagnose“ (http://www.ndr.de/fernsehen/sendungen/visite/media/visite4268.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
02/2010	Management Team - Meeting, Stansted	Research	Europe	10	#1 - #6
02/2010	EU–US Symposia Series, AAAS 2010	Research	Worldwide	5.000	#1 - #19
03/2010	Press Release Berliner Morgenpost; “ Herzpatienten werden oft falsch behandelt“ (http://www.morgenpost.de/printarchiv/wissen/article1273081/Herzpatienten-werden-oft-falsch-behandelt.html)	Higher education	Worldwide	unknown	Coordinating office
04/2010	Exhibition; (Annual Meeting deutsche Gesellschaft für Kardiologie)	Higher education	Germany	500	#1 + #17 + #18 + #16
05/2010	Third Annual Meeting, Varenna	Research	Europe	60	#1 - #19
06/2010	European Atherosclerosis Society Congress, Hamburg	Research	Europe	2.000	#1 - #19
08/2010	Newsletter	Research	Europe	50	Coordinating office
08/2010	European Society of Cardiology Congress, Barcelona	Research	Europe	30.000	#1 - #19
08/2010	Press Release, interview; NDR TV “Bluthochdruck muss behandelt werden “ (http://www.ndr.de/fernsehen/sendungen/visite/chat/chatbluthochdruck104.html)	General Public	Germany	Unknown (TV in Germany, Austria)	Coordinating office
11/2010	Press Release; Universität zu Lübeck „Lübeck als Partnerstandort für neue Deutsche Zentren der Gesundheitsforschung ausgewählt“	Higher education	Germany	unknown	Coordinating office

	http://www.mu-luebeck.de/aktuelles/pressemitteilungen/2011/1110dzgf.php				
12/2010	Newsletter	Research	Europe	50	Coordinating office
12/2010	Press Release, interview; ARTE “Leben ohne Herzinfarkt “ (http://videos.arte.tv/de/videos/leben_ohne_herzinfarkt_-3584224.html)	General Public	Europe	Unknown (TV)	#1 + #2

4.0 Publications of the project

Year 2006 to 2007

1. 1Samani, N.J., J. Erdmann, A.S. Hall, C. Hengstenberg, M. Mangino, B. Mayer, R.J. Dixon, T. Meitinger, P. Braund, H.E. Wichmann, J.H. Barrett, I.R. Konig, S.E. Stevens, S. Szymczak, D.A. Tregouet, M.M. Iles, F. Pahlke, H. Pollard, W. Lieb, F. Cambien, M. Fischer, W. Ouwehand, S. Blankenberg, A.J. Balmforth, A. Baessler, S.G. Ball, T.M. Strom, I. Braenne, C. Gieger, P. Deloukas, M.D. Tobin, A. Ziegler, J.R. Thompson, and H. Schunkert, Genomewide association analysis of coronary artery disease. N Engl J Med, 2007. 357(5): p. 443-53. [IF=55]

2. Lieb W, Mayer B, König IR, Borwitzky I, Götz A, Kain S, Hengstenberg C, Linsel-Nitschke P, Fischer M, Döring A, Wichmann HE, Meitinger T, Kreutz R, Ziegler A, Schunkert H, Erdmann J. Lack of association between the MEF2A gene and myocardial infarction. Circulation. 2008 Jan 15;117(2):185-91. Epub 2007 Dec 17. [IF=11]

3. Aherrahrou Z, Doehring LC, Maria-Ehlers E, Liptau H, Depping R, Linsel-Nitschke P, Kaczmarek PM, Erdmann J, Schunkert H. An alternative splice variant in ABCC6, the gene causing dystrophic calcification, leads to protein deficiency in C3H/He mice. J Biol Chem. 2008 Jan 23; [Epub ahead of print] [IF=6]

4. Schunkert H., Götz A, Peter Braund M.Sc.3, Ralph McGinnis Ph.D4, David-Alexandre Tregouet Ph.D.5, Massimo Mangino Ph.D.3, Patrick Linsel-Nitschke M.D.1, Francois Cambien M.D.5, Christian Hengstenberg M.D.6, Klaus Stark Ph.D.6, Stefan Blankenberg7, Laurence Tiret PhD.5, Pierre Ducimetiere8, Andrew Keniry PhD.4, Mohammed J R Ghori PhD.4, Stefan Schreiber M.D.9, Nour Eddine El Mokhtari M.D.9, Alistair S Hall F.R.C.P.10, Richard J Dixon Ph.D.3, Alison H Goodall Ph.D3, Henrike Liptau M.D.1, Helen Pollard M.Sc3, Daniel F Schwarz M.Sc.2, Ludwig A Hothorn Ph.D.11, H.-Erich Wichmann M.D.12,13, Inke R König Ph.D.2, Marcus Fischer M.D.6, Christa Meisinger M.D.12, Willem Ouwehand F.R.C.Path.14, Cardiogenics Consortium*, Panos Deloukas Ph.D.4, John R Thompson Ph.D.16, Jeanette Erdmann Ph.D.1, Andreas Ziegler Ph.D.2, and Nilesh J Samani FMedSci.3; Repeated Replication and a Prospective Meta-Analysis of the Association Between Chromosome 9p21.3 and Coronary Artery Disease: Circulation (in press) [IF=11]

5. Hunter DJ, Kraft P. Drinking from the fire hose--statistical issues in genomewide association studies. N Engl J Med 2007; 357: 436-439.

6. Rosenzweig A. Scanning the genome for coronary risk. N Engl J Med 2007; 357:
7. 497-499.

8. Kaess B, Fischer M, Baessler A, Stark K, Huber F, Kremer W, Kalbitzer HR, Schunkert H, Riegger G, Hengstenberg C. The lipoprotein subfraction profile: heritability and identification of quantitative trait loci.
   J Lipid Res. 2007 Dec 28; [Epub ahead of print]
   PMID: 18165655 [PubMed - as supplied by publisher]

9. Fischer M, Mayer B, Baessler A, Riegger G, Erdmann J, Hengstenberg C, Schunkert H. Familial aggregation of left main coronary artery disease and future risk of coronary events in asymptomatic siblings of affected patients.
   Eur Heart J. 2007 Oct;28(20):2432-7. Epub 2007 Oct 3.
   PMID: 17914121 [PubMed - in process]

Year 2007 to 2008

1. Fischer M, Mayer B, Baessler A, Riegger G, Erdmann J, Hengstenberg C, Schunkert H., Familial aggregation of left main coronary artery disease and future risk of coronary events in asymptomatic siblings of affected patients, Eur Heart J 2007 Oct, 28(20), 2432-7. [IF = 7,924]

2. Birner CM, Ulucan C, Fredersdorf S, Rihm M, Löwel H, Stritzke J, Schunkert H, Hengstenberg C, Holmer S, Riegger G, Luchner A., Head-to-head comparison of BNP and IL-6 as markers of clinical and experimental heart failure: Superiority of BNP, Cytokine 2007 Nov, 40(2), 89-97. [IF = 2,355]

3. Barbaux S, Tregouet DA, Nicaud V, Poirier O, Perret C, Godefroy T, Francomme C, Combadiere C, Arveiler D, Luc G, Ruidavets JB, Evans AE, Kee F, Morrison C, Tiret L, Brand-Herrmann SM, Cambien F, Polymorphisms in 33 inflammatory genes and risk of myocardial infarction – a system genetics approach, J Mol Med 2007 Nov, 85(11), 1271-80. [IF = 4,82]

4. Wiedmann S, Fischer M, Koehler M, Neureuther K, Riegger G, Doering A, Schunkert H, Hengstenberg C, Baessler A, Genetic variants within the LPIN1 gene, encoding lipin, are influencing phenotypes of the metabolic syndrome in humans, Diabetes 2008 Jan, 57(1), 209-17. [IF = 8,261]

5. Lieb W, Mayer B, König IR, Borwitzky I, Götz A, Kain S, Hengstenberg C, Linsel-Nitschke P, Fischer M, Döring A, Wichmann HE, Meitinger T, Kreutz R, Ziegler A, Schunkert H, Erdmann J, Lack of association between the MEF2A gene and myocardial infarction, Circulation 2008 Jan, 117(2), 185-91. [IF = 12,755]

6. Wallace C, Newhouse SJ, Braund P, Zhang F, Tobin M, Falchi M, Ahmadi K, Dobson RJ, Marçano AC, Hajat C, Burton P, Deloukas P, Brown M, Connell JM, Dominiczak A, Lathrop GM, Webster J, Farrall M, Spector T, Samani NJ, Caulfield MJ, Munroe PB, Genome-wide association study identifies genes for biomarkers of cardiovascular disease: serum urate and dyslipidemia, Am J Hum Genet 2008 Jan;82(1):139-49. [IF = 12,629]

7. Aherrahrou Z, Doehring LC, Ehlers EM, Liptau H, Depping R, Linsel-Nitschke P, Kaczmarek PM, Erdmann J, Schunkert H., An alternative splice variant in Abcc6, the gene causing dystrophic calcification, leads to protein deficiency in C3H/He mice, J Biol Chem 2008 Mar, 283(12), 7608-15. [IF = 5,581]

8. Kaess B, Fischer M, Baessler A, Stark K, Huber F, Kremer W, Kalbitzer HR, Schunkert H, Riegger G, Hengstenberg C, The lipoprotein subfraction profile: heritability and identification of quantitative trait loci, J Lipid Res 2008 Apr, 49(4), 715-23. [IF = 4,336]

9. Linsel-Nitschke P, Schunkert H, Erdmann J, Congestive heart failure is a common disease with complex inheritance – new perspectives through genome wide association studies, Internist (Berl) 2008 Apr, 49(4), 405-10, 412. [IF = 0,271]

10. Frey UH, Lieb W, Erdmann J, Savidou D, Heusch G, Leineweber K, Jakob H, Hense HW, Löwel H, Brockmeyer NH, Schunkert H, Siffert W, Characterization of the GNAQ promoter and association of increased Gq expression with cardiac hypertrophy in humans, Eur Heart J 2008 Apr, 29(7), 888-97. [IF = 7,924]

11. Schunkert H, Götz A, Braund P, McGinnis R, Tregouet DA, Mangino M, Linsel-Nitschke P, Cambien F, Hengstenberg C, Stark K, Blankenberg S, Tiret L, Ducimetiere P, Keniry A, Ghori MJ, Schreiber S, El Mokhtari NE, Hall AS, Dixon RJ, Goodall AH, Liptau H, Pollard H, Schwarz DF, Hothorn LA, Wichmann HE, König IR, Fischer M, Meisinger C, Ouwehand W, Deloukas P, Thompson JR, Erdmann J, Ziegler A, Samani NJ; Cardiogenics Consortium: Repeated replication and a prospective meta-analysis of the association between chromosome 9p21.3 and coronary artery disease, Circulation 2008 Apr, 117(13), 1675-84. [IF = 12,755]

12. Stark K, Reinhard W, Neureuther K, Wiedmann S, Sedlacek K, Baessler A, Fischer M, Weber S, Kaess B, Erdmann J, Schunkert H, Hengstenberg C, Association of common polymorphisms in GLUT9 gene with gout but not with coronary artery disease in a large case-control study, PLoS ONE 2008 Apr, 3(4), 1948.

13. Brouilette SW, Whittaker A, Stevens SE, van der Harst P, Goodall AH, Samani NJ, Telomere length is shorter in healthy offspring of subjects with coronary artery disease: support for the telomere hypothesis, Heart 2008 Apr, 94(4), 422-5. [IF = 3,708]

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