SUPPLEMENTAL MATERIAL

Optical Coherence Tomography Findings in Patients with Coronary Stent Thrombosis

A report of the PREvention of late Stent Thrombosis by an Interdisciplinary Global European effort (PRESTIGE) consortium

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Supplementary methods

OCT quantitative analysis

Metallic stent struts were identified as bright, signal-intense structures with blooming and dorsal shadowing: polymeric bioresorbable stent struts appear as a “black box” area surrounded by bright reflecting frames without abluminal shadowing.[1] The first and last analyzed frame at the stented segment was defined as the OCT frame allowing the drawing of a complete circumference using the strut contour, where struts were present in at least 3⁄4 of the perimeter. Distal and proximal reference measurements were performed in none or minimally diseased cross-sections within 10 mm from the stent edges. For morphometric analysis, standard definitions of cross-sectional area (CSA) and diameter were applied as previously reported.[2] Stent and lumen CSA were measured throughout the entire length of the stent. Lumen area was not assessed in presence of remaining thrombus obscuring the luminal border in at least one third of the luminal circumference. Mean reference area was calculated as the sum of the distal and proximal non-stented reference lumen area divided by 2. In case the pullback did not include analyzable distal and proximal non-stented reference segments, the reference area was derived from the proximal and distal most stented segment. Stent expansion index was calculated as minimum stent area divided by mean reference area. Inter-observer variability for core lab morphometric measurements was assessed by repeat analysis of 10 patients performed by a second operator.

Presence of thrombus, stent strut coverage, stent strut apposition, inter-strut cavities, degree and type of neointimal tissue characteristics consistent with neoatherosclerosis were evaluated at frame level. Thrombus was defined as intraluminal protruding mass with irregular borders with or without adherence to stent struts or luminal tissue. The greatest

longitudinal thrombus extent was calculated using the number of consecutive frames with any thrombus. Strut coverage was adjudicated on a frame-level basis. Struts were considered uncovered if any part of the strut was visibly exposed to the lumen. Conversely, struts covered by visible thrombus were classified as thrombus covered struts and counted separately in the analysis. The number of consecutive frames with uncovered struts was counted and the greatest longitudinal extent of uncovered struts was measured. Malapposition was considered present when the axial distance between the luminal surface of the strut to the lumen contour was greater than the strut thickness (including polymer, if present) including a correction factor to account for strut blooming artifact.[2] Readers blinded to stent type performed the assessment of malapposition. After finishing all measurements, stent type was unblinded and appropriate cut-off values were used to determine coverage and malapposition for each patient. Distance of malapposition was derived from the distance between the luminal surface of the strut and the lumen contour. Maximum malapposition distance and area of malapposition were recorded. The maximum length of malapposition was derived by the number of consecutive frames with malapposed struts. Inter-strut cavities (or coronary in-stent evaginations) were defined as the presence of an outward bulge in the luminal vessel contour between apposed struts with a maximum depth of the bulge greater than 1/3 of the lumen diameter. Atherosclerotic changes of the neointima (neoatherosclerosis) were defined by the presence of one or more of the following: lipid laden tissue within the stent, defined as a signal-poor region with diffuse border and light signal attenuation, possibly masking deep strut detection; thin-cap fibroatheroma (TCFA), defined as plaque with lipid-laden tissue with a fibrous cap thickness ≤ 65 µm at the thinnest measured point, or neointimal calcification, characterized by a signal-poor region with sharp demarcation within the overlying neointima [3-5]. In the core lab

analysis, neoatherosclerosis was adjudicated when lipid-laden tissue or TCFA involved more than 50% of the analyzable arc at the involved cross-section.

Supplementary results

Comparison of patients with and without OCT imaging

In a sensitivity analysis comparing patients with ST with analyzable OCT imaging who were included in the present analysis (n=217) versus those without analyzable OCT imaging (n=458) we found was no significant difference in the proportion of patients with acute/subacute or late/very late ST 62/217 (28.6%) versus 150/455 (33.0%) and 155/217 (71.4%) versus 305/455 (67.0%) respectively; P=0.25).

Baseline characteristics of patients with and without OCT imaging are shown in **Supplementary Table 2**.

OCT core laboratory analysis

Mean difference between methods and within-patient standard deviation for proximal lumen area, distal lumen area and minimal stent area were 1.03 and 1.43 mm^2, 0.02 and 0.20 mm^2, -0.24 and 0.27 mm^2, respectively. For these parameters, repeatability coefficients and intra-class correlation coefficients (Shrout and Fleiss) were 3.96 and 0.62, 0.57 and 0.99, 0.76 and 0.97 respectively.

Supplementary Table 1. Results of platelet reactivity testing

	Early stent thrombosis	Late/very late stent thrombosis	p-value
Platelet reactivity with VerifyNow $P2Y_{12}$ (n=49)			
High platelet reactivity	15/17 (88.2%)	22/32 (68.8%)	0.18
Platelet reactivity units	257±86	245±86	0.64
Platelet reactivity with Multiplate adenosine diphosphate (ADP) (n=51)			
High platelet reactivity	15/20 (75.0%)	19/31 (61.3%)	0.31
Platelet reactivity units (U)	76±46	63±33	0.25

Data are shown as n (%) or mean ± standard deviation

Supplementary Table 2. Baseline characteristics of patients presenting with stent thrombosis with and without analyzable OCT imaging

	With OCT imaging (N= 217)	Without OCT imaging (N= 458)	p-value
Age (years)	63.7±11.9	64.1±11.8	0.69
Male	178/217 (82.0%)	374/458 (81.7%)	0.91
Diabetes	54/217 (24.9%)	105/455 (19.4%)	0.61
insulin dependent	8/214 (12.9%)	41/435 (9.4%)	0.15
Hypercholesterolemia	181/193 (93.8%)	376/420 (89.5%)	0.09
Arterial hypertension	99/211 (46.9%)	229/451 (50.8%)	0.36
Active smoker	62/210 (29.5%)	123/448 (27.5%)	0.58
Severely impaired left ventricular function*	9/217 (4.2%)	15/451 (3.3%)	0.59
Prior bypass operation	14/217 (6.5%)	35/455 (7.7%)	0.56
Prior myocardial infarction	100/217 (46.1%)	303/427 (71.0%)	<0.001
Renal insufficiency	9/217 (4.2%)	19/453 (4.2%)	0.98
Clinical presentation			0.29
ST-elevation myocardial infarction	158/217 (72.8%)	361/458 (78.8%)	
Non-ST-elevation myocardial infarction	46/217 (21.2%)	79/458 (17.3%)	
Unstable angina	10/217 (4.6%)	12/458 (2.6%)	
Culprit vessel			
Left main only	2/214 (0.9%)	9/456 (2.0%)	0.53
LAD only	90/214 (42.1%)	177/456 (38.8%)	0.42
LCx only	29/214 (13.5%)	56/456 (12.3%)	0.64
RCA only	78/214 (36.5%)	182/456 (39.9%)	0.39
…….LAD and LCx	6/214 (2.8%)	3/456 (0.7%)	0.07
…….LAD and RCA	2/214 (0.9%)	2/456 (0.4%)	0.77
…….LCx and RCA	3/214 (1.4%)	1/456 (0.2%)	0.20
Saphenous vein graft	4/214 (1.9%)	26/456 (5.7%)	0.03
Bifurcation lesion	38/205 (18.5%)	76/438 (17.4%)	0.71

Number of stents implanted in vessel with stent thrombosis	333	685	
Stent type at index procedure			
- bare metal stent	110/333 (33.0%)	187/685 (27.3%)	0.07
- early generation DES	45/333 (1.0%)	120/685 (17.5%)	0.12
- newer generation DES	163/333 (48.9%)	316/685 (46.1%)	0.44
- bioresorbable DES	6/333 (1.8%)	11/685 (1.6%)	>0.99
- unknown	9/333 (2.7%)	51/685 (7.4%)	0.003
Stent diameter (mm)	3.2±1.4	3.1±0.5	0.62
Stent length (mm)	19.8±6.3	20.6±7.7	0.12
TIMI flow at presentation			0.70
0/1	168/212 (79.3%)	370/451 (82.0%)	
2	21/212 (9.9%)	33/451 (12.6%)	
3	23/212 (13.1%)	48/451 (9.9%)	

Data are shown as n (%) or mean ± standard deviation

*severely impaired left ventricular function was defined as ejection fraction <30%

Supplementary Table 3: Estimated probabilities for findings according to time interval between index stenting and stent thrombosis at a frame-level adjusted by generalized linear mixed models

Predicted average probability* (95% CI) for a frame to have any*	Acute stent thrombosis	Subacute stent thrombosis	Late stent thrombosis	Very late stent thrombosis
Uncovered Struts	77.4% (56.4%; 90.0%)	57.0% (43.2%; 69.8%)	22.4% (11.1%; 39.9%)	5.3% (3.6%; 7.6%)
Thrombus Covered Struts	17.3% (7.3%; 35.6%)	24.5% (15.7%; 36.1%)	3.8% (1.5%; 9.3%)	3.2% (2.1%; 4.9%)
Uncovered or Thrombus-Covered Struts	99.3% (96.1%; 99.9%)	96.6% (92.4%; 98.5%)	34.3% (15.0%; 60.7%)	9.6% (6.2%; 14.5%)
Malapposed Struts	21.8% (8.4%; 45.6%)	8.5% (4.6%; 15.3%)	6.7% (2.5%; 16.3%)	2.0% (1.2%; 3.3%)
Struts with Neoatherosclerosis	0.0% (0.0%; 0.0%)	0.0% (0.0%; 0.0%)	0.0% (0.0%; 0.0%)	2.8% (1.3%; 5.9%)
Interstrut Cavities	0.0% (0.0%; 0.0%)	0.1% (0.0%; 0.3%)	1.2% (0.3%; 4.5%)	0.7% (0.3%; 1.4%)

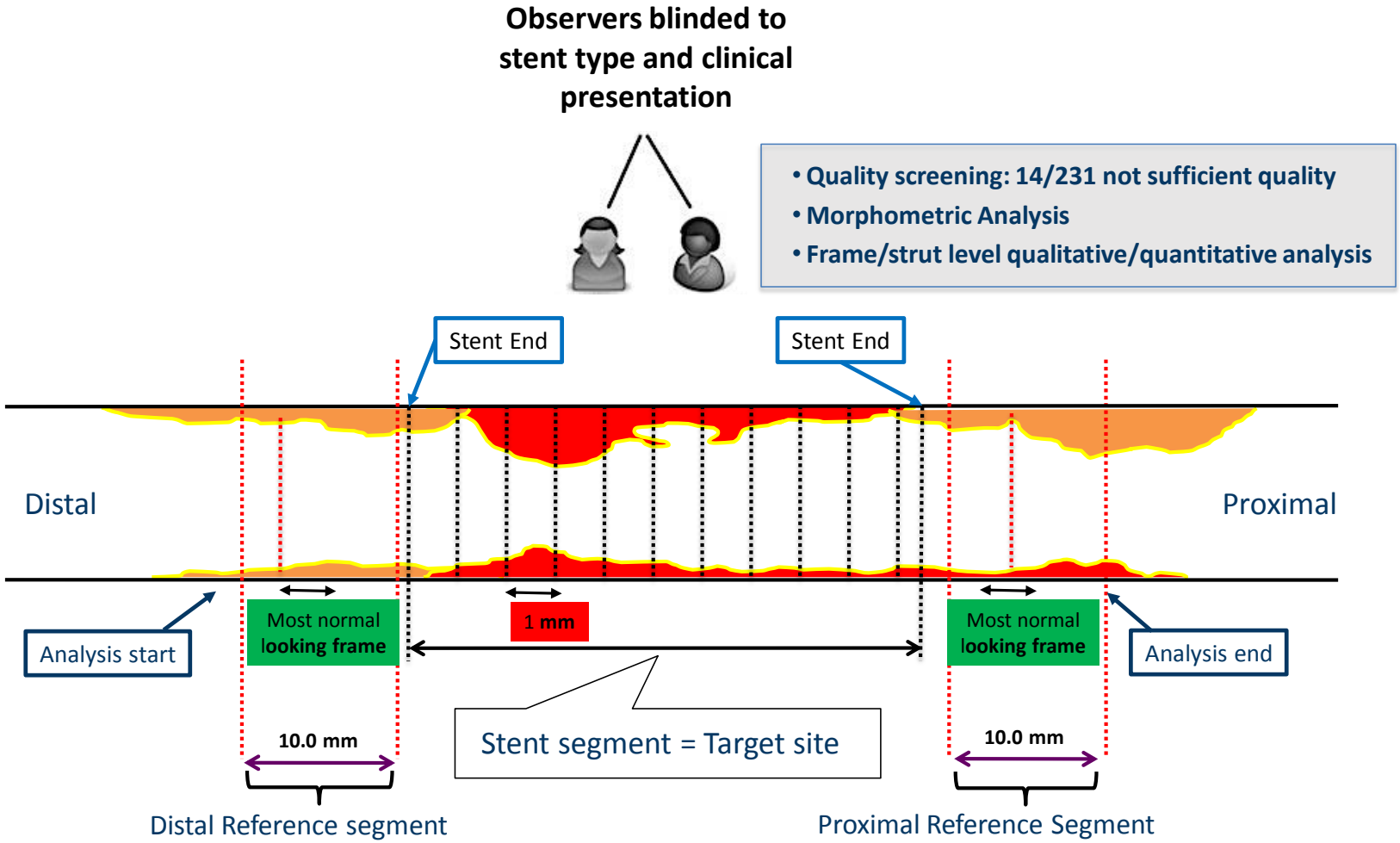
Estimates were obtained using a mixed effects logistic regression model, including a fixed effect for time and a random intercept per patient

* Predicted probability for an 'average' patient, i.e. with random intercept of zero

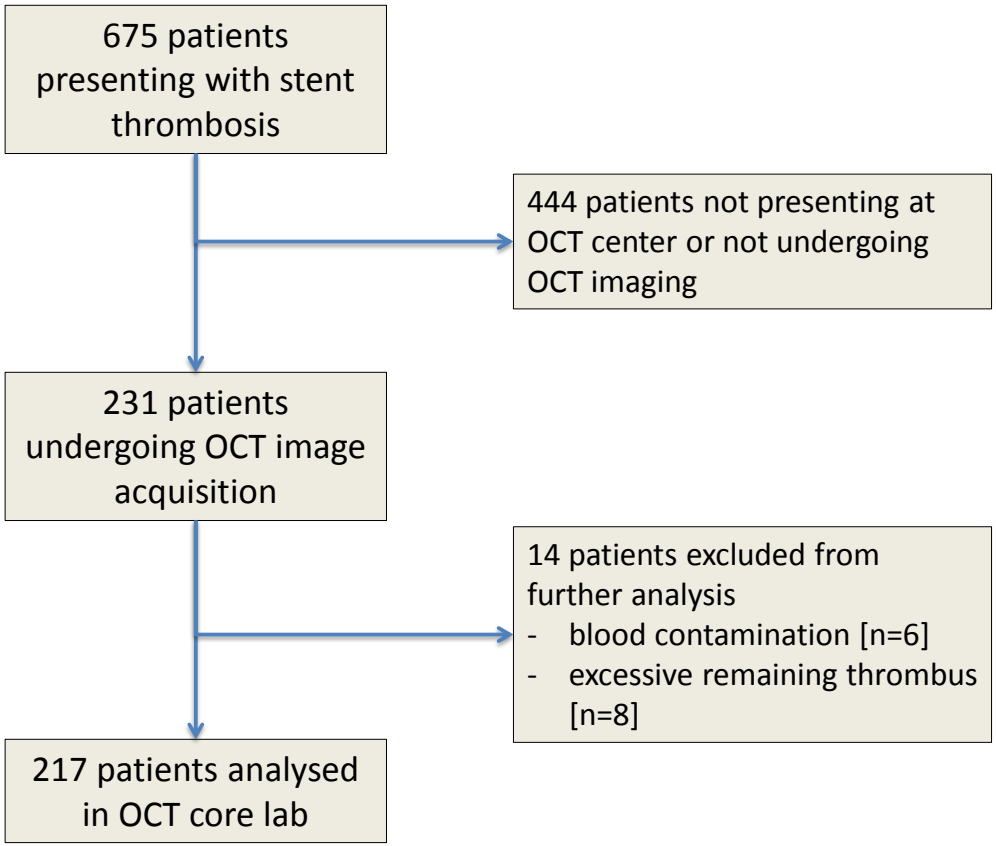
Supplementary Table 4: Results of imaging adjudication committee analysis for contributory findings in stent thrombosis

	Acute stent thrombosis (N=15)	Subacute stent thrombosis (N=47)	Late stent thrombosis (N=21)	Very late stent thrombosis (N=134)
Uncovered struts	15 (100%)	43 (91.5%)	13 (61.9%)	65/134 (48.5%)
Malapposed struts	9 (60%)	24 (51.1%)	8 (38.1%)	37/134 (27.6%)
Underexpansion	3 (20%)	31 (66.0%)	8 (38.1%)	35/134 (26.1%)
Severe restenosis	0 (0%)	0 (0%)	8 (38.1%)	43/134 (32.1%)
Neoatherosclerosis	0 (0%)	0 (0%)	0 (0%)	52/134 (38.8%)
Neoatherosclerosis with rupture	0 (0%)	0 (0%)	0 (0%)	31/134 (23.1%)
Inter strut cavities	0 (0%)	1 (2.1%)	5 (23.8%)	18/134 (13.4%)
Distal edge dissection	1 (6.7%)	3 (6.4%)	0 (0%)	1/134 (0.8%)
Proximal edge dissection	0 (0%)	0 (0%)	0 (0%)	1/134 (0.8%)
Edge segment disease & plaque rupture	2 (13.3%)	5 (10.6%)	4 (19.1%)	16/134 (11.9%)
Stent overlap	3 (20.0%)	13 (27.7%)	5 (23.8%)	33/134 (24.6%)
Stent fracture	0 (0%)	1 (2.1%)	0 (0%)	1/134 (0.8%)

Supplementary Figure 1. Outline of work flow of core lab analysis



Supplementary Figure 2. Study flow chart



OCT = optical coherence tomography

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Appendix A: General description of the PRESTIGE Consortium

In 2010, a consortium of 14 European institutions joined forces to investigate stent thrombosis. The project consortium was named PRESTIGE—PREvention of late Stent Thrombosis by an Interdisciplinary Global European effort—and was coordinated by the Deutsches Herzzentrum München in Munich, Germany. It has run for 4 years, 2010–14. The project was funded by the European Commission under the Seventh Framework Programme (Grant Agreement No.: HEALTH-F2-2010-260309). Within the project, the scientists want to develop new concepts to identify and prevent ST. The strategy includes a basic scientific approach to decrypt the molecular and cellular mechanisms underlying ST, a bio-engineering approach focused on the development and testing of new intravascular imaging tools and stent materials to prevent stent thrombosis, plus a clinically-orientated effort to better characterize the burden of ST across Europe.

The consortium is led by Prof. Adnan Kastrati and Prof. Steffen Massberg and has participants from nine European Union countries, involving both academic centres and small- and medium-sized companies.

PRESTIGE Consortium

Partners: Deutsches Herzzentrum München (DHM); Azienda Ospedaliera Papa Giovanni XXIII (BER); Samodzielny Publiczny Zaklad Opieki Zdrowotnej Szpital Uniwersytecki W Krakowie (KRAK); St. Antonius Ziekenhuis Nieuwegein (NIE); University of Leicester (ULEIC); Universitäts-Herzzentrum Freiburg-Bad Krozingen GmbH (UHZ); Institut national de la santé et de la recherche médicale (INSERM); Rigas Tehniska Universitate (RTU); Kitozyme S.A. (KIZ); Helmholtz Zentrum München, Deutsches Forschungszentrum für Gesundheit und Umwelt GmbH (HMGU); Katholieke Universiteit Leuven (K.U.LEUVEN); Servicio Madrileño de Salud: Hospital Universitario Clinico San Carlos (SC) and Hospital Universitario de La Princesa (HULP); BIOTRONIK SE & Co. KG (BIO); neoplas GmbH (NEO).

Investigators: ***Belgium***: Tom Adriaenssens (K.U.LEUVEN), Emanuele Barbato, (Cardiovascular Center, Aalst), Ian Buysschaert (ZNA Middelheim), Mickaël Chausson (initially KIZ, now Synolyne Pharma), Dries De Cock (K.U.LEUVEN), Jo Dens (Oost-Limburg Hospital, Genk), Walter Desmet (K.U.Leuven), Sandrine Gautier (initially KIZ, now Synolyne Pharma), Paul Vermeersch (ZNA Middelheim), Peter Sinnaeve (K.U.LEUVEN); ***Czech Republic***: Ota Hlinomaz (ICRC, St. Anne University Hospital, Brno), Ladislav Groch (ICRC, St. Anne University Hospital, Brno), Jan Sitar (ICRC, St. Anne University Hospital, Brno), Michal Rezek (ICRC, St. Anne University Hospital, Brno), Jiri Semenka (ICRC, St. Anne University Hospital, Brno), Martin Novak (ICRC, St. Anne University Hospital, Brno), Jiri Sikora (ICRC, St. Anne University Hospital, Brno); ***France***: Helene Abergel (INSERM), Jeremie Abtan (INSERM), Pierre Aubry (INSERM), Gregory Ducrocq (INSERM), Laurent Feldman (INSERM), Eric Garbarz (INSERM), Dominique Himbert (INSERM), Martine Jandrot-Perrus (INSERM), Jean-Michel Juliard (INSERM), Didier Letourneur (INSERM), Pierre Mangin (INSERM), Mohammed Nejjari (INSERM), Véronique Olivier (INSERM), Caroline Roques (INSERM), Emmanuel Sorbets (INSERM), Ph. Gabriel Steg (INSERM), Marina Urena-Alcazar (INSERM); ***Germany***: Robert A. Byrne (DHM), Sue Chandraratne (initially DHM, later Klinikum der Universität München), Matthias Gratz (BIO); Michael Joner (DHM), Adnan Kastrati (DHM), Elisabeth Kennerknecht (DHM), Ildiko Konrad (DHM), Tobias Koppara (DHM), Steffen Massberg (initially DHM, later Klinikum der Universität München), Franz-Josef Neumann (UHZ), Vasilis Ntziachristos (HMGU), Sheryl Opinaldo (initially DHM, later Klinikum der Universität München), Vanessa Philippi (initially DHM, later Klinikum der Universität München), Julia Riegger (initially DHM, later Klinikum der Universität München), Amir Rosenthal (HMGU), Alexander Rzany (BIO), Christian Schulz (initially DHM, later Klinikum der Universität München), Kristin Steigerwald (DHM), Tomohiso Tada (DHM), Anna Titova (initially DHM, later Klinikum der Universität München), Dietmar Trenk (UHZ), Christian Valina (UHZ), Andreas Vogelsang (NEO), Erion Xhepa (DHM); ***Italy***: Chiara Bernelli (BER); Micol Coccato (BER), Giulio Guagliumi (BER), Kenichi

Komukai (BER), Vasile Sirbu (BER); ***Latvia***: Garry Kerch (RTU); ***The Netherlands***: Giovanni Amoroso (Onze Lieve Vrouwe Gasthuis, Amsterdam), Jurriën ten Berg (NIE), Willem J.M. Dewilde (Amphia Ziekenhuis, Breda), Thea C. Godschalk (NIE), Antonius A.C.M. Heestermans (Noordwest Ziekenhuisgroep, Alkmaar), Darshni A. Jhagroe (NIE), Joanne J. Wykrzykowska (Academisch Medisch Centrum, Amsterdam), Mark H.M. Winkens (TweeSteden ziekenhuis, Tilburg); ***Poland***: Dariusz Dudek (KRAK), Łukasz Rzeszutko (KRAK), Roman Wojdyla (KRAK), Wojciech Zasada (KRAK); ***Spain***: Fernando Alfonso (HULP, SC), Javier Cuesta (HULP), Miguel Medina (SC); ***United Kingdom***: Colin Berry (University of Glasgow; Golden Jubilee National Hospital, Glasgow), James Cotton (The Royal Wolverhampton Hospitals NHS Trust), Nick Curzen (University Hospital Southampton NHS Foundation Trust), Margaret McEntegart (Golden Jubilee National Hospital, Glasgow), Robert Gerber (East Sussex Healthcare NHS Trust), Anthony Gershlick (ULEIC), Alison H. Goodall (ULEIC), Simon Hetherington (Kettering General Hospital NHS Foundation Trust), Jonathan Hill (King's College Hospital NHS Foundation Trust), Damian Kelly (Derby Hospitals NHS Foundation Trust), Nikesh Malik (ULEIC), Keith Oldroyd (Golden Jubilee National Hospital, Glasgow), Helen Routledge (Worcestershire Acute Hospitals NHS Trust), Joanne Shannon (Frimley Health Foundation Trust), Venkatesan Suresh (Plymouth Hospitals NHS Trust), Azfar Zahman (Newcastle Upon Tyne Hospitals NHS Foundation Trust).

<u>Work Packages:</u>

Work package 1 (WP1):	Gaining a better mechanistic understanding of the molecular and cellular events triggering late ST Leader: Martine Jandrot-Perrus (INSERM), Co-Leader: Steffen Massberg (initially DHM, later Klinikum der Universität München)
Work package 2 (WP2):	Developing and validating novel strategies to reduce late ST

	Leader: Michael Joner (DHM), Co-Leader: Didier Letourneur (INSERM)
Work package 3(WP3):	Developing and evaluating novel imaging technologies Leader: Giulio Guagliumi (BER), Co-Leader: Vasilis Ntziachristos (HMGU)
Work package 4 (WP4):	Performing a multi-stranded characterisation of patients with late ST Leader: Walter Desmet (K.U.LEUVEN), Co-Leader: Anthony Gershlick (ULEIC)
OCT imaging adjudication committee:	This group was comprised of experts with documented expertise in clinical and preclinical OCT use and evaluation of coronary stents. Members: Tom Adriaenssens (K.U.LEUVEN), Takashi Akasaka (Wakayama University, Japan), Fernando Alfonso (SC), Robert A. Byrne (DHM), Giulio Guagliumi (BER), Michael Joner (DHM), Vasile Sirbu (BER).
OCT core lab:	Tom Adriaenssens (K.U.LEUVEN), Robert A. Byrne (DHM), Dries De Cock (K.U.LEUVEN), Thea Godschalk (NIE), Kenichi Komukai (BER), Nikesh Malik (ULEIC), Tomohisa Tada (DHM); Erion Xhepa (DHM)
Thrombus analysis core lab:	Sue Chandraratne, Steffen Massberg, Sheryl Opinaldo, Vanessa Philippi, Julia Riegger, Christian Schulz, Anna Titova (all initially DHM, later Klinikum der Universität München).