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PLATINUM QCA: a prospective, multicentre study assessing clinical, angiographic, and intravascular ultrasound outcomes with the novel platinum chromium thin-strut PROMUS Element everolimus-eluting stent in *de novo* coronary stenoses

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KEYWORDS

- coronary artery disease
- intravascular ultrasound
- PROMUS Element
- quantitative coronary angiography
- restenosis

Abstract

Aims: Assess clinical, angiographic, and intravascular ultrasound results in lesions treated with the PROMUS Element platinum chromium everolimus-eluting stent (EES).

Methods and results: Patients (N=100) with one *de novo* target lesion ≤34 mm long and reference vessel diameter (RVD) ≥2.25–≤4.25 mm were enrolled at 14 sites. The primary endpoint was the 30-day composite of cardiac death, myocardial infarction, target lesion revascularisation (TLR), or definite/probable stent thrombosis (ST). The efficacy endpoint of 9-month in-stent late loss in workhorse lesions (defined as RVD ≥2.5–≤4.25 mm, lesion ≤24 mm) was compared to a performance goal based on historical results with TAXUS Express paclitaxel-eluting stents. Post-procedure incomplete stent apposition (ISA) was compared to a performance goal based on results with the PROMUS/XIENCE V EES in SPIRIT III. Mean age was 61.8±9.9 years; 77.0% were male; 19% had medically treated diabetes. Baseline RVD was 2.72±0.53 mm; lesion length was 15.4±7.0 mm. The primary endpoint occurred in one patient (periprocedural ST with TLR) with no additional major clinical events through one year. Nine-month in-stent late loss in workhorse lesions (0.17±0.25 mm, N=73) and post-procedure ISA (5.7%, 5/88) were below performance goals ($p<0.001$).

Conclusions: Through one year, PROMUS Element EES had an acceptable safety and efficacy profile with low in-stent late loss and post-procedure ISA.

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Introduction

While a clear restenosis advantage of coronary drug-eluting stents (DES) over bare-metal stents (BMS) has been demonstrated in randomised, controlled trials (RCT), about 7-10% of patients treated with DES still require repeat revascularisation[1-4]. Furthermore, first generation DES have shown higher rates of very late stent thrombosis (ST) compared with BMS[5]. PROMUS® (XIENCE V®), a second-generation everolimus-eluting stent (EES), has shown reduced angiographic late loss, target lesion revascularisation (TLR), myocardial infarction (MI), and ST compared to paclitaxel-eluting stents[6-9].

Despite these improvements in clinical outcomes, there is still a need for better performing stents. In the PROMUS Element™ EES, the same drug and polymer coating used in PROMUS is applied to a novel platinum chromium stent platform designed to improve deliverability, radiopacity, radial strength and recoil [10,11]. In a rabbit endothelial cell denudation model, the thinner strut bare-metal Element stent exhibited more rapid strut coverage and endothelialisation than Liberté or Express[12], and in a non-injured porcine coronary revascularisation model, PROMUS Element demonstrated vascular compatibility equivalent to PROMUS (XIENCE V)[13]. On the basis of these promising preclinical data, PLATINUM QCA (Prospective, Randomised, MuLticenter TriAl To Assess an EverolImus-Eluting CoroNary Stent System [PROMUS EleMent] Quantitative Coronary Angiography), a prospective, single-arm, multicentre observational study, was designed to evaluate the clinical, angiographic and intravascular ultrasound (IVUS) outcomes in lesions treated with the PROMUS Element stent.

Methods

DEVICE DESCRIPTION

The PROMUS Element EES (Boston Scientific Corporation, Natick, MA, USA [BSC]) has been described previously[11]. In brief, the thin-strut, balloon-expandable stent consists of a novel platinum chromium alloy[10] with the antiproliferative agent everolimus[14] (100 µg/cm^2) applied in a durable, biocompatible acrylic polymer and fluorinated copolymer[15] identical to that used in the PROMUS (XIENCE V) EES[3] (manufactured and distributed by Abbott Vascular, Santa Clara, CA, USA, as XIENCE V and distributed by BSC as PROMUS). PROMUS Element and PROMUS (XIENCE V) are shown in **Figure 1**.

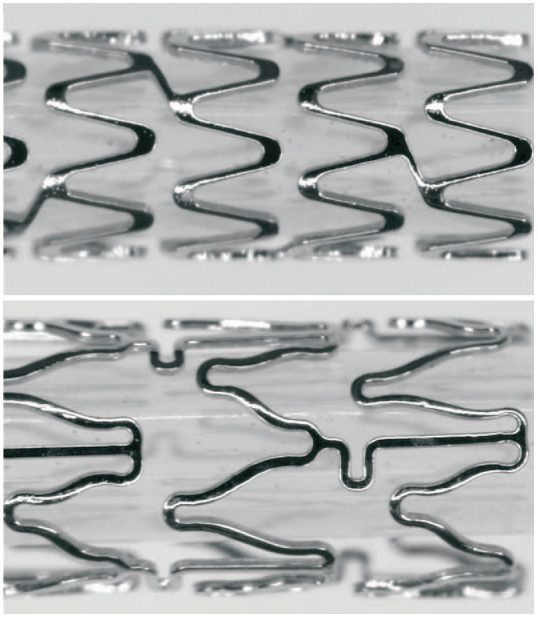

Figure 1. *PROMUS Element above and PROMUS (XIENCE V) everolimus-eluting stents.*

STUDY DESIGN AND PROCEDURE

Enrolled patients presented with stable or unstable angina pectoris or documented silent ischaemia and were to be treated with one stent for a single *de novo* target lesion ≤34 mm long with a visually estimated diameter stenosis ≥50% to <100% in an artery with a reference vessel diameter (RVD) of ≥2.25 mm to ≤4.25 mm. Treatment of one non-target lesion in a non-target vessel with a commercial treatment (e.g., stent, balloon angioplasty, excluding brachytherapy) was also allowed provided it occurred before target lesion intervention and was a clinical and angiographic success (defined as visually assessed mean lesion diameter stenosis <50% [<30% for stents] with TIMI 3 flow without prolonged chest pain or MI). Staged or planned revascularisations following the index procedure were prohibited. Patients with myocardial infarction within 72 hours prior to the index procedure and elevated cardiac markers at the time of the procedure were excluded from enrolment. Complete inclusion and exclusion criteria are provided in Appendix A. Clinical follow-up was scheduled at 1, 9, and 12 months along with 9-month angiographic and IVUS follow-up. Dual antiplatelet therapy (aspirin and a thienopyridine) was mandated to reduce the risk of thrombosis. Thienopyridine treatment (clopidogrel, ticlopidine, or prasugrel in accordance with approved country-specific labelling) was required for at least six months (12 months or longer recommended); aspirin was required indefinitely.

The Ethics Committee at each participating centre approved the study protocol. Informed written consent prior to enrolment or the performance of any study-specific procedures or tests was required from all patients. An independent clinical events committee (CEC) adjudicated all reported events of death, MI, target vessel revascularisation (TVR) and ST, and an independent data monitoring committee provided oversight of aggregate safety data. Angiograms were evaluated by an independent angiographic core laboratory (Beth Israel Deaconess Medical Center, Boston, MA, USA) using software from Medis Medical Imaging Systems (Leiden, The Netherlands). Intravascular ultrasound data were evaluated at an independent core laboratory (MedStar Research Institute, Washington, D.C., USA). Additional PLATINUM study organisation and oversight committee membership are provided in Appendix B. The study is registered at www.clinicaltrials.gov, identifier NCT00824434.

Endpoints

The primary endpoint was the 30-day composite rate of cardiac death, MI, TLR, or ST defined as definite or probable per the Academic Research Consortium [ARC] definitions[18]. Stent thrombosis was included in the primary endpoint given the concern about ST in the interventional community. Additional endpoints included technical and clinical procedural success (defined in Appendix C); death, MI, revascularisation, and ST at each follow-up period; and QCA and IVUS analyses at nine months. The efficacy endpoints, 9-month in-stent late loss by QCA in workhorse lesions (defined as RVD ≥2.5 mm and ≤4.25 mm and lesion length ≤24 mm by visual estimate) and post-procedure incomplete stent apposition (ISA) by IVUS, were compared to pre-specified performance goals (see "Statistical methods"). Incomplete stent apposition was defined as separation of one or more stent struts from the vessel wall with evidence of blood speckles behind the stent strut on IVUS. Additional endpoint definitions are provided in Appendix C.

STATISTICAL METHODS

The efficacy endpoints of 9-month in-stent late loss (in workhorse lesions) and post-procedure ISA were compared to predefined performance goals (PG). For 9-month in-stent late loss, the PG of 0.44 mm (0.41 mm + delta [0.03 mm]) was based on historical results in similar lesions treated with the TAXUS Express stent in the TAXUS IV and TAXUS V studies and the data were compared using a one group t-test ($p<0.05$ for significance). Assuming an expected in-stent late loss of 0.18±0.50 mm and a sample of 60 workhorse patients, the study had 99% power to conclude in-stent late loss was less than the PG. This conclusion would be made if the one-sided upper 95% confidence bound on the observed value was below the PG. For post-procedure ISA, the PG (34.4%) was based on historical PROMUS (XIENCE V) post-procedure ISA data from the SPIRIT III study,[7] and the data were compared using a one-group exact test ($p<0.05$ for significance). Assuming an expected ISA rate of 17.2% and a sample of 70 patients, the study had 95% power to conclude the ISA rate was below the PG if the one-sided Clopper-Pearson exact upper 95% confidence bound on the observed value was less than the PG. No statistical testing was done for the additional endpoints. Patient, lesion, and procedural characteristics and event rates were analysed using descriptive statistics; simple proportions with 95% confidence intervals were used for categorical variables with continuous data provided as mean ± standard deviation. All statistical analyses were performed using SAS version 8 or higher (SAS Institute, Inc., Cary, NC, USA).

Results

PATIENT, LESION, AND PROCEDURAL CHARACTERISTICS

The PLATINUM QCA study enrolled 100 patients at 14 sites in Australia, Malaysia, New Zealand, and Singapore (Appendix D) from March to July 2009. Baseline demographics and lesion characteristics are shown in **Table 1**. The cohort included 85 workhorse, 12 long-lesion (RVD ≥2.25-≤4.25 mm, lesion >24-≤34 mm), and three small-vessel (RVD ≥2.25-<2.5 mm, lesion ≤28 mm) target lesions (by visual estimate). Procedural outcomes appear in **Table 2**. Device technical success, defined as successful delivery and deployment of the study stent to the target lesion without balloon rupture or embolisation was 100%.

Table 1. Baseline clinical and lesion characteristics.

Characteristic	All patients (N=100)
Patient	
Male (%)	77.0 (77)
Age (yr)	61.8±9.9 (100)
Cardiac history	
Stable angina (%)	57.0 (57)
Unstable angina (%)	38.0 (38)
Silent ischaemia (%)	2.0 (2)
Left ventricular ejection fraction (%)[a]	64.4±13.3
Previous myocardial infarction (%)	39.0 (39)
Previous percutaneous coronary intervention (%)	31.0 (31)
Previous coronary artery bypass graft (%)	5.0 (5)
Cardiac risk factors	
Current smoking (%)	15.2 (15)
Diabetes, medically treated (%)[b]	19.0 (19)
Hypertension (%)[c]	66.0 (66)
Hyperlipidaemia (%)[c]	81.8 (81)
Family history of coronary artery disease (%)	52.7 (49)
Clopidogrel treatment[d]	
Prior regimen/loading dose (%)	98.0 (98)
Discharge (%)	100.0 (100)
Lesion (by QCA)	
Reference vessel diameter (mm)	2.72±0.53
Minimum lumen diameter (mm)	0.71±0.34
Lesion length (mm)	15.40±7.03
Diameter stenosis (%)	74.09±10.93

Data are %(n) or mean±SD; [a] N=97; [b] Insulin and/or oral medication; [c] Requiring medication; [d] All patients (N=100) had a prior regimen/loading dose of aspirin; no patients received ticlopidine or prasugrel; QCA: quantitative coronary angiography

Table 2. Procedural outcomes.

Parameter	Value
Technical success[a]	100% (108)
Clinical procedural success[b]	99.0% (99)
Max stent deployment pressure (atm)	14.08±2.55 (100)
Pre-dilatation used	100.0% (100)
Post-dilatation used	91.0% (91)
Max post-dilatation pressure (atm)	18.01±3.55 (91)

Data are %(n) or mean±SD (n); [a] Defined as successful delivery and deployment of the study stent to the target vessel without balloon rupture or stent embolisation; N=108 stents; [b] Defined as mean lesion diameter stenosis <30% with TIMI 3 flow with no in-hospital myocardial infarction, target vessel revascularisation, or cardiac death; N=100 patients

CLINICAL OUTCOMES AT 30 DAYS AND ONE YEAR

Clinical follow-up was 100% at 30 days and one year; outcomes are shown in **Table 3**. The primary endpoint, the 30-day composite rate of cardiac death, MI, TLR or ST, occurred in one patient (1.0%) who had a periprocedural ST with TLR and a non-target lesion TVR. There were no additional major clinical events through one year. The single patient with an event was a 62-year-old female with a previous triple bypass graft who received a 3.5x12 mm stent implanted in the proximal left anterior descending artery (LAD). One day after the index procedure the patient had chest pain and anterior ST-segment elevation which improved after treatment with heparin, nitroglycerine, and tirofiban. Angiography showed a widely patent stent, but there was a slight haziness suggestive of thrombus at the origin of an adjacent branch vessel. There was also severe vessel spasm in the distal LAD. Assessment by IVUS showed stent malapposition and further balloon angioplasty of the stent was performed. The maximum CK and CK-MB after the event were 328 U/L (upper limit of normal [ULN] 190 U/L) and 13 U/L (ULN 10 U/L).

QUANTITATIVE CORONARY ANGIOGRAPHY

Follow-up at 9-months was 88% for QCA. Nine-month in-stent late loss in workhorse lesions was 0.17±0.25 mm (N=73) with an upper confidence bound of 0.22 mm, significantly below the pre-specified performance goal ($p<0.001$). **Figure 2** shows cumulative frequency distribution curves for in-stent percent diameter stenosis (DS) and in-stent minimum lumen diameter (MLD) in workhorse lesions pre-procedure, post-procedure, and at follow-up. The mean pre-procedure DS of 72.95±10.63% was reduced to 3.57±8.32% after stent implantation and remained low at 9.34±10.44% by nine months. Mean MLD was 0.75±0.34 mm before stent implantation, 2.68±0.44 mm post-procedure, and 2.50±0.46 mm at follow-up.

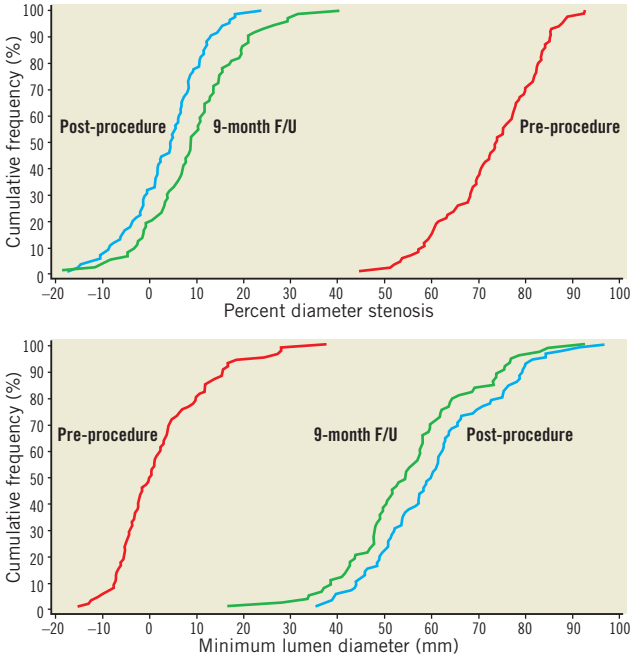

Figure 2. *Cumulative frequency distribution curves in workhorse lesions; red: pre-procedure, blue: post-procedure, green: 9-month follow-up. In-stent percent diameter stenosis (above); In-stent minimum lumen diameter.*

Table 4 shows QCA results in all lesion types (N=88). At nine months, in-stent late loss was 0.20±0.28 mm and binary restenosis was 1.1% (N=1).

INTRAVASCULAR ULTRASOUND

Post-procedure ISA was present in 5.7% of patients (5/88, upper confidence bound of 11.6%), significantly less than the predeter-

Table 3. Clinical outcomes at 30 days and one year.

Outcome	30 days (N=100)	1 year (N=100)
Primary endpoint (composite of cardiac death, MI, TLR, ST)[a]	1.0% (1)	–
All death, MI, or TVR	1.0% (1)	1.0% (1)
All death	0.0% (0)	0.0% (0)
MI	0.0% (0)	0.0% (0)
Q-wave MI	0.0% (0)	0.0% (0)
Non–Q-wave MI	0.0% (0)	0.0% (0)
TVR	1.0% (1)	1.0% (1)
TLR	1.0% (1)	1.0% (1)
TLF[b]	1.0% (1)	1.0% (1)
ST[c]	1.0% (1)	1.0% (1)

Data are binary rates,%(n); [a] Periprocedural ST with TLR and revascularisation of a non-target lesion in the target vessel; [b] Defined as ischaemia-driven TLR, or MI/cardiac death related to the target vessel; [c] Per Academic Research Consortium definitions definite/probable; MI: myocardial infarction; ST: stent thrombosis; TLF: target lesion failure; TLR: target lesion revascularisation; TVR: target vessel revascularisation

Table 4. Quantitative coronary angiography outcomes.

Parameter (N=88)[a]	Post-procedure	9 months
Minimum lumen diameter, in-stent (mm)	2.64±0.46	2.44±0.49
Minimum lumen diameter, in-segment[b] (mm)	2.27±0.52	2.20±0.49
Acute gain, in-stent (mm)	1.93±0.47	–
Acute gain, in-segment (mm)	1.56±0.51	–
Percent diameter stenosis, in-stent	3.58±7.98	10.0±11.59
Percent diameter stenosis, in-segment	17.99±7.88	19.66±8.95
Late loss, in-stent (mm)	–	0.20±0.28
Late loss, in-segment (mm)	–	0.07±0.27
Binary restenosis, in-stent	–	1.1% (1)
Binary restenosis, in-segment	–	1.1% (1)

Data are mean±SD or % (n); post-procedure reference vessel diameter= 2.76±0.53 mm; [a] Among the 12 patients excluded from analysis, three did not have angiographic analysis, eight had angiographic follow-up out-of-window (120-300 days post-procedure), and one was ineligible for angiographic follow-up due to a periprocedural target lesion revascularisation; [b] Stented segment plus 5 mm at the proximal and distal edges

Table 5. Intravascular ultrasound analysis.

Parameter	Post-procedure[a]	9 months
Stent volume (mm^3)	186.83±89.61 (88)	196.29±102.10 (73)
Lumen volume (mm^3)	186.61±89.32 (88)	183.56±98.72 (73)
Vessel volume (mm^3)	356.17±173.26 (77)	378.19±191.63 (71)
Neointimal volume (mm^3)	0.50±1.62 (88)	12.73±11.74 (73)
Net volume obstruction in-stent (%)	0.28±0.86 (88)	7.24±6.22 (73)
Incomplete stent apposition (%)	5.7% (5/88)	0.0% (0/73)
Paired analysis (%)[b]	5.8% (4/69)	0.0% (0/69)

Numbers are mean ±SD (N) or% (n/N); [a] 12 patients had unreadable post-procedure analyses; [b] Includes patients with both 9-month and post-procedure assessments; among the 31 patients excluded from paired analysis, four had unreadable post-procedure and 10 had unreadable follow-up IVUS analyses, six did not have follow-up IVUS analysis, six had IVUS follow-up out-of-window (120-300 days post-procedure), IVUS follow-up was not available for analysis in four, and one was ineligible for IVUS follow-up due to a periprocedural target lesion revascularisation; IVUS: intravascular ultrasound

mined performance goal of 34.4% reported for PROMUS (XIENCE V) in the SPIRIT III trial (p<0.001)[7]. Follow-up at 9-months was 83% for IVUS. Among patients with paired analyses (N=69), all post-procedure ISA was resolved at nine months with no late acquired ISA. Percent volume obstruction in 73 lesions was 7.2±6.2 at nine months. Additional IVUS outcomes are shown in **Table 5**.

Discussion

This prospective multicentre study evaluated the clinical, angiographic, and intravascular ultrasound outcomes in *de novo* coronary stenoses treated with the thin-strut, platinum chromium PROMUS Element everolimus-eluting stent. The primary endpoint (30-day composite of cardiac death, MI, TLR, or ST) occurred in 1.0% of patients (1/100, with one patient having a periprocedural ST with TLR). There were no additional major cardiac events through one year. The performance goals for the angiographic and IVUS efficacy measures were met as in-stent late loss in workhorse lesions (RVD ≥2.5–≤4.25 mm, lesion length ≤24 mm; N=73) was 0.17±0.25 mm compared to a value of 0.44 mm based on historical TAXUS Express results (P<0.001)[2,19] and post-procedure ISA (N=88) was 5.7% versus 34.4% for PROMUS (XIENCE V) in SPIRIT III (p<0.001)[7]. These outcomes suggest that the everolimus/polymer combination in PROMUS (XIENCE V) can be successfully transferred to the platinum chromium Element stent platform.

The mean in-stent late loss of 0.17 mm at nine months with PROMUS Element in workhorse lesions is comparable to that previously reported for PROMUS (XIENCE V) in the SPIRIT First trial (0.10±0.21 mm at six months[3] and 0.24±0.27 mm at one year[20]), SPIRIT II (0.11±0.27 mm at six months[6]), and SPIRIT III (0.16 ± 0.41 mm at eight months[7]). Late loss in-segment at nine months with PROMUS Element in all lesions was 0.07±0.27 mm (N=88). This result was also similar to that reported for in-segment late loss with PROMUS (XIENCE V) in SPIRIT First (0.07±0.19 mm [six months][3]; 0.14±0.24 mm [one year][20]), SPIRIT II (0.07±0.33 mm [six months][6]), and SPIRIT III (0.14±0.41 mm [eight months][7]).

Intravascular ultrasound outcomes at nine months in PLATINUM QCA were similar to outcomes reported for PROMUS (XIENCE V) in the SPIRIT trials. Neointimal hyperplasia was 12.7±11.7 mm^3 and percent volume obstruction was 7.2±6.2 (N=73) with PROMUS Element compared to 10.1±11.5 mm^3 and 6.9±6.4% (N=101), respectively, for PROMUS (XIENCE V) at eight months in SPIRIT III[7]. Similar outcomes were reported at six months in SPIRIT First (10±13 mm^3 and 8.0±10.4%, N=21)[3] and SPIRIT II (4±7 mm^3 and 2.5±4.7%, N=100)[6].

Post-procedure ISA with PROMUS Element (5.7%, 5/88) was significantly less than that reported for PROMUS (XIENCE V) in SPIRIT III (34.4%, p<0.001). This may reflect differences in stent design, but could also be related to other factors including the extensive use (91.0%) of post-dilatation and/or the maximum post-dilatation balloon pressure (18.0±3.6 atm) in the PLATINUM QCA study. Among SPIRIT III patients with both post-procedure and 8-month IVUS follow-up (110 patients, 117 lesions) the post-dilatation rate was 48.7% with 15.7±3.3 atm maximum post-dilatation balloon pressure. In this patient subset, post-procedure ISA was 33.3%, which was similar to that observed in the overall SPIRIT III population but significantly greater than the ISA rate observed in the SPIRIT III Japan registry (15.9%, p=0.006; 73 patients with 82 lesions).[21] Post-dilatation rates and maximum post-dilatation balloon pressure were higher in the SPIRIT III Japan registry (65.9% and 17.9±2.7 atm, respectively).[21]

Importantly, among patients with paired IVUS analyses there were no occurrences of late acquired ISA with PROMUS Element (0/69, **Table 5**). Indeed, late ISA was not present in any patient treated with PROMUS Element (0/73). The absence of late acquired ISA is consistent with that observed for PROMUS (XIENCE V) (1.1% at eight months in SPIRIT III[7], none at six months in SPIRIT II[6], none at six months or one year in SPIRIT First[20]). Also of note, there was no significant change in vessel dimensions and volume in the PROMUS Element stented segment indicating the absence of positive vessel remodelling due to chronic inflammation.

Study limitations

PLATINUM QCA was a small, first human use, non-randomised study with comparisons to performance goals based on historical results, and there is a potential for bias due to differences in patient complexity or treatment patterns. Operators may also have modified their treatment strategy based on their knowledge of the previ-

ously observed rates of post-procedure incomplete apposition. The study was not powered to investigate clinical endpoints; in the PLATINUM Workhorse RCT, a companion trial in the PLATINUM Clinical Trial Program with 1,530 enrolled patients, PROMUS Element was found non-inferior to PROMUS for 12-month target lesion failure and the two stents had similar, low 12-month rates of cardiac death, MI, TLR, and ST[22]. Finally, results obtained in PLATINUM QCA may not apply to patient and lesion types excluded from enrolment.

Conclusions

Through one year the PROMUS Element everolimus-eluting stent has an acceptable safety and efficacy profile with low in-stent late loss and post-procedure incomplete stent apposition. These results suggest that the drug and polymer combination used in PROMUS (XIENCE V) can successfully be transferred to the platinum chromium Element stent platform.

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Funding source

Boston Scientific Corporation, Natick, MA, USA

Conflict of interest statement

Prof. Meredith has served as a consultant for BSC, Abbott Vascular, and Medtronic. Dr. Stone is on the Scientific Advisory Board for, and has received honoraria from BSC and Abbott Vascular. Dr. Teirstein reports receiving research funding, consulting fees and honorarium from BSC, Medtronic, Cordis, and Abbott. Drs. Allocco, Dawkins, and Starzyk are full-time employees and stockholders of BSC. The other authors have no conflicts of interest to declare.

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Online data supplement

Appendix A. Patient and angiographic inclusion and exclusion criteria.

Appendix B. PLATINUM QCA study organisation and processes.

Appendix C. Definitions.

Appendix D. PLATINUM QCA investigative sites.

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Online appendices

Appendix A. Patient and angiographic inclusion and exclusion criteria

Clinical inclusion criteria	• Patient must be at least 18 years of age • Patient (or legal guardian) understands the study requirements and the treatment procedures and provides written informed consent before any study-specific tests or procedures are performed • For patients less than 20 years of age enrolled at a Japanese site, the patient and the patient's legal representative must provide written informed consent before any study-specific tests or procedures are performed • Patient is eligible for PCI • Patient has documented stable angina pectoris or documented silent ischaemia; or unstable angina pectoris • Patient is an acceptable candidate for CABG • Patient has LVEF ≥30% as measured within 30 days prior to enrolment • Patient is willing to comply with all protocol-required follow-up evaluations
Angiographic inclusion criteria (visual estimate)	• Target lesion must be a de novo lesion located in a native coronary artery with a visually estimated RVD ≥2.25 mm and ≤4.25 mm • Target lesion length must measure (by visual estimate) ≤34 mm • Target lesion must be in a major coronary artery or branch with visually estimated stenosis ≥50% and <100% with TIMI flow >1.
Clinical exclusion criteria	• Patient has clinical symptoms and/or ECG changes consistent with acute MI • Patient has had a known diagnosis of recent MI (i.e., within 72 hours prior to the index procedure) and has elevated enzymes at the time of the index procedure as follows. ○ Patients are excluded if any of the following criteria are met at the time of the index procedure. ▪ If CK-MB >2× upper limit of normal (ULN), the patient is excluded regardless of the CK Total. ▪ If CK-MB is 1-2× ULN, the patient is excluded if the CK Total is >2× ULN. ▪ If CK Total/CK-MB are not used and Troponin is, patients are excluded if the following criterion is met at the time of the index procedure. ○ Troponin >1× ULN with at least one of the following. ▪ Patient has ischaemic symptoms and ECG changes indicative of ongoing ischaemia (e.g., >1 mm ST segment elevation or depression in consecutive leads or new left bundle branch block [LBBB]); ▪ Development of pathological Q-waves in the ECG; or ▪ Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. ***Note*:** For patients with unstable angina or patients who have had a recent MI, CK Total/CK-MB (or Troponin if CK Total/CK-MB are not used) must be documented prior to enrolling/randomising the patient. • Patient has received an organ transplant or is on a waiting list for an organ transplant • Patient is receiving or scheduled to receive chemotherapy within 30 days before or after the index procedure • Patient is receiving oral or intravenous immunosuppressive therapy (i.e., inhaled steroids are not excluded) or has known life-limiting immunosuppressive or

Appendix A. Patient and angiographic inclusion and exclusion criteria

autoimmune disease (e.g., human immunodeficiency virus, systemic lupus erythematosus, but not including diabetes mellitus)

- Patient is receiving chronic (≥72 hours) anticoagulation therapy (e.g., heparin, coumadin) for indications other than acute coronary syndrome
- Patient has a platelet count <100,000 cells/mm^3 or >700,000 cells/mm^3
- Patient has a white blood cell (WBC) count <3,000 cells/mm^3
- Patient has documented or suspected liver disease, including laboratory evidence of hepatitis
- Patient is on dialysis or has known renal insufficiency (i.e., estimated creatinine clearance <50 ml/min by the Cockcroft Gault formula, or [(140-age)*lean body weight (in kg)]/[plasma creatinine (mg/dl)*72])
- Patient has a history of bleeding diathesis or coagulopathy or will refuse blood transfusions
- Patient has had a cerebrovascular accident or transient ischaemic attack within the past 6 months, or has any permanent neurologic defect that may cause non-compliance with the protocol
- Target vessel(s) or side branch has been treated with any type of PCI (e.g., balloon angioplasty, stent, cutting balloon, atherectomy) within 12 months prior to the index procedure
- Target vessel(s) has been treated within 10 mm proximal or distal to the target lesion (by visual estimate) with any type of PCI (e.g., balloon angioplasty, stent, cutting balloon, atherectomy) at any time prior to the index procedure
- Non-target vessel or side branch has been treated with any type of PCI (e.g., balloon angioplasty, stent, cutting balloon, atherectomy) within 24 hours prior to the index procedure
- Planned or actual target vessel(s) treatment with an unapproved device, directional or rotational coronary atherectomy, laser, cutting balloon, or transluminal extraction catheter immediately prior to stent placement
- Planned PCI or CABG after the index procedure
- Patient previously treated at any time with coronary intravascular brachytherapy
- Patient has a known allergy to the study stent system or protocol-required concomitant medications (e.g., stainless steel, platinum, cobalt, chromium, nickel, tungsten, acrylic, fluoropolymers, everolimus, thienopyridines, aspirin, contrast) that cannot be adequately premedicated
- Patient has an active peptic ulcer or active gastrointestinal bleeding
- Patient has one of the following.
  - Other serious medical illness (e.g., cancer, congestive heart failure) that may reduce life expectancy to less than 24 months
  - Current problems with substance abuse (e.g., alcohol, cocaine, heroin, etc.)
  - Planned procedure that may cause non-compliance with the protocol or confound data interpretation
- Patient is participating in another investigational drug or device clinical trial that has not reached its primary endpoint
- Patient intends to participate in another investigational drug or device clinical trial within 12 months after the index procedure
- Patient with known intention to procreate within 12 months after the index procedure (Women of child-bearing potential who are sexually active must agree to use a

Appendix A. Patient and angiographic inclusion and exclusion criteria

	reliable method of contraception from the time of screening through 12 months after the index procedure.) • Patient is a woman who is pregnant or nursing (A pregnancy test must be performed within 7 days prior to the index procedure in women of child-bearing potential.) • Patient has more than 1 target lesion, or more than 1 target lesion and 1 non-target lesion, which will be treated during the index procedure
Angiographic Exclusion Criteria (visual estimate)	• Target lesion meets any of the following criteria: ○ Aorto-ostial location (i.e., lesion located within 5 mm of the ostium by visual estimate) ○ Left main location ○ Located within 5 mm of the origin of the left anterior descending coronary artery or left circumflex coronary artery by visual estimate ○ Located within a saphenous vein graft or an arterial graft ○ Will be accessed via a saphenous vein graft or an arterial graft ○ Involves a side branch ≥2.0 mm in diameter by visual estimate ○ Involves a clinically significant side branch <2.0 mm in diameter by visual estimate that has a clinically significant stenosis at the ostium ○ TIMI flow 0 (total occlusion) or TIMI flow 1 prior to wire crossing ○ Excessive tortuosity proximal to or within the lesion ○ Extreme angulation proximal to or within the lesion ○ Target lesion and/or target vessel proximal to the target lesion is moderately to severely calcified by visual estimate ○ Restenotic from previous intervention ○ Thrombus, or possible thrombus, present in the target vessel • Non-target lesion to be treated during the index procedure meets any of the following criteria: ○ Located within the target vessel ○ Located within a bypass graft (venous or arterial) ○ Left main location ○ Chronic total occlusion ○ Involves a complex bifurcation (e.g., bifurcations requiring treatment with more than 1 stent) ○ Restenotic from previous intervention • Patient has unprotected left main coronary artery disease (>50% diameter stenosis) • Patient has protected left main coronary artery disease and a target lesion in the LAD or LCX • Patient has an additional clinically significant lesion(s) in the target vessel for which an intervention within 12 months after the index procedure is likely to be required ***Note:*** Multiple focal stenoses will be considered as a single lesion if they can be completely covered with 1 stent.

Abbreviations: CABG=coronary artery bypass graft; CK=creatine kinase; CK-MB= creatine kinase-myoglobin band; ECG=electrocardiogram; LAD=left anterior descending artery; LBBB=left bundle branch block; LCX=left circumflex artery; LVEF=left ventricular ejection fraction; MI=myocardial infarction; PCI=percutaneous coronary intervention; RVD= reference vessel diameter; TIMI=thrombolysis in myocardial infarction; ULN=upper limit of normal

Appendix B: PLATINUM QCA Study Organisation and Processes

Sponsor	Boston Scientific Corporation, Natick, MA, USA
Principal Investigator	IT Meredith, Monash Medical Centre, Clayton, Victoria, Australia
Clinical Events Committee	DG Hurrell (Chair, Minneapolis Cardiology Associations, Minneapolis, MN, USA); J Chambers (Metropolitan Cardiology Consultants, P.A., Minneapolis, MN, USA); D.D. Laxson (Minnesota Heart Clinic, P.A., Edina, MN, USA); YL Wang (Cardiovascular/Peripheral Vascular Interventions Minneapolis Heart Institute, Minneapolis, MN, USA); RF Wilson (University of Minnesota Cardiovascular Division, Minneapolis, MN, USA)
Data Monitoring Committee	WD Weaver (Chair, Henry Ford Heart and Vascular Institute, Detroit, MI, USA); SR Bailey (University of Texas, San Antonio, TX, USA); DP Faxon (Brigham and Women's Hospital, Boston, MA, USA); DJ Moliterno (University of Kentucky Medical Center, Lexington KY, USA); JGP Tijssen (DMC Statistician, University of Amsterdam, Amsterdam, The Netherlands); A Greenbaum (DMC Medical Liaison, Henry Ford Hospital, Detroit, MI, USA)
Steering Committee	GW Stone (Chair, Columbia University Medical Center and The Cardiovascular Research Foundation, New York, NY, USA); PS Teirstein (Scripps Clinic, Division of Cardiovascular Diseases, La Jolla, CA, USA); IT Meredith (MonashHEART, Southern Health, Monash Medical Centre, Clayton, Victoria, Australia); K Dawkins, E Rose, D Allocco, P Maurer (all Boston Scientific Corporation, Natick, MA, USA)
Angiography Core Laboratory	Beth Israel Deaconess Medical Center, Boston, MA, USA; Jeffrey J. Popma (Director)
Intravascular Ultrasound Core Laboratory	MedStar Research Institute, Washington, D.C., USA; Neil J. Weissman (Vice President, Research Programs)
Device Management	Biomedical Research Institute of New Mexico, Albuquerque, NM, USA
Electronic Data Capture	Medidata, New York, NY, USA
Image Management Services	BIOCLINICA, INC., Newtown, PA, USA
Biostatistical Analysis	Boston Scientific: P Lam (Director); P Pereda (Manager); E McMullen (Principal Biostatistician)
Clinical Project Management	Boston Scientific: E Rose (Vice President, Global Project Management); J Maffeo (Global Program Manager); L Bussone (Program Manager, PLATINUM QCA)
Data Management	Boston Scientific: C Muza (Director); V de Medeiros (Principal Clinical Data Manager)
Medical Monitor and Safety Monitoring	Boston Scientific: D Allocco (Director); L Giannini (Director); M Sondhi (Director); B Hennessey (Safety Trial Manager)

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Appendix C. Definitions.

Cardiac death	Death due to any of the following: • Acute MI • Cardiac perforation/pericardial tamponade • Arrhythmia or conduction abnormality • CVA through hospital discharge or CVA suspected of being related to the procedure • Death due to complication of the procedure, including bleeding, vascular repair, transfusion reaction, or bypass surgery • Any death in which a cardiac cause cannot be excluded Non-cardiac death is defined as a death not due to cardiac causes as defined above.
Clinical angiographic success (non-target lesion)	Mean lesion diameter stenosis <50% (<30% for stents) in 2 near-orthogonal projections with TIMI 3 flow, as visually assessed by the physician, without the occurrence of prolonged chest pain or ECG changes consistent with MI.
Clinical procedural success	Mean lesion diameter stenosis <30% in 2 near-orthogonal projections with TIMI 3 flow, as visually assessed by the physician, without the occurrence of in-hospital MI, TVR, or cardiac death.
Myocardial infarction	Periprocedural MI (occurring within 48 hours of the index procedure or any repeat revascularisation): • Q-Wave MI: Development of new (i.e., not present on the patient's ECG before allocation) pathological Q-waves in 2 or more leads lasting ≥0.04 seconds with post-procedure CK-MB levels elevated above normal. If the only enzyme available is Troponin, it must be >ULN and the baseline level must have been <ULN. • Non–Q-Wave MI: De novo elevation of CK Total levels >3× ULN without the presence of new Q-waves (i.e., not present on the patient's ECG before allocation). If CK-MB is performed, it must be positive. In the absence of either CK or CK MB, Troponin may be used and must be >3× ULN and the baseline level must have been <ULN. There must also be any one of the following: ECG changes indicative of new ischaemia (new ST-T changes or LBBB), imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality. Perioperative MI (for patients undergoing bypass surgery): • Total CK MB >5× ULN. If no CK MB is available, Troponin may be used. It must be >5× ULN, the baseline level must have been <ULN, and there must be evidence of any one of the following: new LBBB or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium, -OR-

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	• Presence of new pathologic Q-waves as defined above. Spontaneous MI: • Q-Wave MI: Development of new (i.e., not present on the patient's ECG before allocation) pathological Q-waves in 2 or more leads lasting ≥0.04 seconds with post-procedure CK MB levels elevated above normal. If the only enzyme available is Troponin, it must be >ULN and the baseline level must have been <ULN. • Non–Q-Wave MI: De novo elevation of CK Total levels >2× ULN without the presence of new Q-waves (i.e., not present on the patient's ECG before allocation). If CK MB is performed, it must be positive. In the absence of either CK or CK MB, Troponin may be used and must be >2× ULN and the baseline level must have been <ULN. There must also be any one of the following: ECG changes indicative of new ischaemia (new ST-T changes or LBBB), or imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality.
Non-target lesion	A lesion for which treatment with a study stent is not attempted. **Note:** Multiple focal stenoses will be considered as a single lesion if they can be completely covered with one stent.
Stent thrombosis	Definite or probable according to the ARC definitions[18]
Target lesion	A lesion that meets the angiographic selection criteria and is to be treated with a study stent during the index procedure. **Note:** Multiple focal stenoses will be considered as a single lesion if they can be completely covered with one stent.
Target lesion failure	Any ischemia-driven revascularisation of the target lesion, MI (Q-wave and non–Q-wave) related to the target vessel, or (cardiac) death related to the target vessel. If it cannot be determined with certainty whether the MI or death was related to the target vessel, it will be considered a TLF.
Target lesion revascularisation	Any ischemia-driven repeat percutaneous intervention to improve blood flow of the successfully treated target lesion or bypass surgery of the target vessel with a graft distal to the successfully treated target lesion. A TLR will be considered ischemia-driven if the target lesion diameter stenosis is ≥50% by QCA and there is presence of clinical or functional ischemia which cannot be explained by other coronary or graft lesions. Clinical or functional ischemia is any of the following: • The patient has a positive functional study corresponding to the area served by the target lesion. • The patient has ischemic ECG changes at rest in a distribution consistent with the target vessel. • The patient has ischemic symptoms referable to the target lesion. A TLR will be considered as ischemia-driven if the lesion diameter stenosis is ≥70% by QCA even in the absence of clinical or functional ischemia.
Target vessel	*Any ischemia-driven revascularisation of the target vessel, MI (Q-wave and non–Q-wave) related to the target vessel, or death related to the target vessel. For the*

Appendix C. Definitions.

Failure	*purposes of this protocol, if it cannot be determined with certainty whether the MI or death was related to the target vessel, it will be considered a TVF.*
Target vessel revascularisation	Defined as a TLR (see above for definition) or a TVR remote (defined below): Any ischemia-driven repeat percutaneous intervention to improve blood flow or bypass surgery of not previously existing lesions with diameter stenosis ≥50% by QCA in the target vessel, excluding the target lesion. A TVR will be considered ischaemia-driven if the target vessel diameter stenosis is ≥50% by QCA and any of the following are present: • The patient has a positive functional study corresponding to the area served by the target vessel. • The patient has ischaemic ECG changes at rest in a distribution consistent with the target vessel. • The patient has ischaemic symptoms referable to the target vessel. A TVR will also be considered as ischaemia-driven if the lesion diameter stenosis is ≥70% even in the absence of clinical or functional ischaemia.
Technical success	Successful delivery and deployment of the study stent to the target vessel, without balloon rupture or stent embolisation

ARC: Academic Research Consortium; CK: creatine kinase; CK-MB: creatine kinase-myoglobin band; CVA: cerebrovascular accident; ECG: electrocardiogram; LBBB; left bundle branch block; MI: myocardial infarction; QCA: quantitative coronary angiography; TIMI: thrombolysis in myocardial infarction; TLF: target lesion failure; TLR: target lesion revascularisation; TVF: target vessel failure; TVR: target vessel revascularisation; ULN: upper limit of normal

Appendix D. PLATINUM QCA investigative sites.

Principal investigator	Site name and address
Ian Meredith, Study PI	Monash Medical Centre, Clayton, Victoria, Australia
Dougal McClean	Christchurch Hospital, Christchurch, Canterbury, New Zealand
Gerry Wilkins	Dunedin Hospital, Dunedin, Otago, New Zealand
Alan Whelan	Freemantle Hospital, Freemantle, WA, Australia
Robaayah Zambahari	Institut Jantung Negara, Kuala Lumpur, Malaysia
Craig Juergens	Liverpool Hospital, Liverpool, NSW, Australia
Douglas Scott	Middlemore Hospital, Centre for Clinical Research and Effective Practice, Otahuhu, Auckland, New Zealand
Aaron Wong	National Heart Centre Singapore, Singapore
Seif El-Jack	North Shore Hospital, Takapuna, Auckland, New Zealand
Steve Worthley	Royal Adelaide Hospital, Adelaide, SA, Australia
Michael Muhlmann	Sir Charles Gairdner Hospital, Nedlands, Perth, WA, Australia
Robert Whitbourn	St. Vincent's Hospital, Fitzroy, Victoria, Australia
Darren Walters	The Prince Charles Hospital, Brisbaine, Queensland, Australia
Scott Harding	Wellington Hospital, Newtown, Wellington, New Zealand

PI: principal investigator