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Antiplatelet De-Escalation After Percutaneous Coronary Intervention: Bleeding Reduction Without Ischaemic Cost

Yannis Papaharalambous[1*], Wilhelmina Steenkamp-Oude[2], Cristiano Melandri[3]

[1]*Department of Interventional Cardiology, Kyrenia Heart Centre, Paphos, Cyprus*
[2]*Centre for Evidence Synthesis, Randstad Heart Institute, The Hague, Netherlands*
[3]*Department of Pharmacy, Friulian Institute of Cardiovascular Medicine, Trieste, Italy*

*Correspondence to: Yannis Papaharalambous, Department of Interventional Cardiology, Kyrenia Heart Centre, Paphos, Cyprus
E-mail: y.papaharalambous@kyrenia-hc.cy

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Introduction

Potent P2Y12 inhibition with ticagrelor or prasugrel, added to aspirin after percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS), lowers ischaemic risk more than clopidogrel does, and that advantage is exactly why both drugs became the guideline default. The same potency that blocks a future thrombotic event also blocks the platelet response needed to stop a bleed, and the bleeding cost is front-loaded: it is highest in the weeks after PCI, precisely when the ischaemic benefit of potent inhibition is thought to matter least, because the culprit lesion has already been treated and the residual thrombotic risk is falling.

De-escalation strategies were built to exploit that mismatch: they keep potent inhibition for the highest-risk early period, then step down to a gentler regimen once the acute thrombotic risk has receded. Four distinct versions of this idea have now been tested in adequately powered randomised trials -- an unguided switch to clopidogrel at a fixed timepoint, an unguided reduction in prasugrel dose at a fixed timepoint, a platelet-function-guided switch keyed to an individual patient's measured on-treatment reactivity, and a CYP2C19 genotype-guided initial drug choice -- and each has reported a bleeding benefit.

What is less settled is whether that bleeding benefit comes free, or whether it is bought, in at least some strategies or some patients, at the cost of a real but statistically undetected rise in ischaemic events. This review reads TROPICAL-ACS, TOPIC, HOST-REDUCE-POLYTECH-ACS, POPular Genetics, and TALOS-AMI alongside a pooled meta-analysis and two risk-stratified subgroup analyses to ask that question directly, rather than to repeat the reassuring headline that each trial's own abstract offers.

Material and Methods

This is a narrative review, not a systematic review with a pre-registered protocol. It draws on the primary publications of five randomised de-escalation trials in ACS patients undergoing PCI, a meta-analysis pooling those and related trials, a post hoc complex-PCI subgroup analysis, and a post hoc atherothrombotic-risk subgroup analysis, identified through the sources cited here and citation tracking between them. All percentages, hazard ratios, confidence intervals, and p-values reported are taken directly from the cited sources; none has been invented, estimated, or extrapolated. The trials differ deliberately in the de-escalation strategy tested -- unguided switching, unguided dose reduction, platelet-function-guided switching, and genotype-guided initial selection -- so that guided and unguided approaches can be compared directly rather than assumed to behave identically.

Results

Platelet-function-guided de-escalation: TROPICAL-ACS

TROPICAL-ACS randomised 2610 ACS patients treated with PCI and initially loaded with prasugrel to either continued standard prasugrel or a guided de-escalation strategy: one week of prasugrel, one week of clopidogrel, then platelet function testing at day 14 to confirm adequate inhibition before continuing clopidogrel, with a switch back to prasugrel if reactivity was insufficient [1]. At 1 year, the combined ischaemic-and-bleeding primary endpoint occurred in 7.3% of the guided de-escalation group versus 9.0% of the standard group (hazard ratio [HR] 0.81, 95% CI 0.62-1.06), meeting the pre-specified non-inferiority margin (p=0.0004) without reaching superiority (p=0.12) [1]. Figure 1 sets this result alongside the other four trials reviewed here.

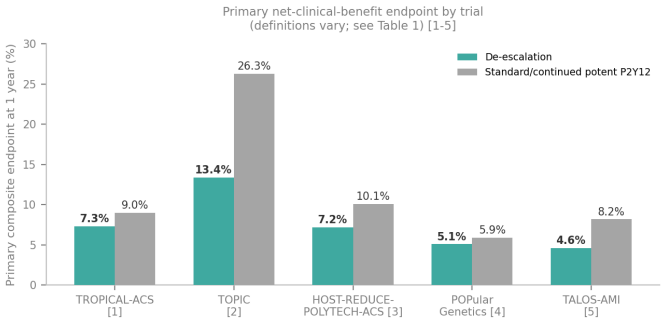

Figure 1: Primary composite endpoint at 1 year, de-escalation versus comparator, across the five trials reviewed. Endpoint definitions differ by trial; see Table 1 [1-5].

The ischaemic components of the TROPICAL-ACS primary endpoint were essentially unchanged between arms: all-cause mortality occurred in 1.0% of both groups (12 versus 11 events, p=0.85) and definite stent thrombosis in 0.2% of both groups (3 versus 2 events, p=0.66) [1]. Because the trial's own bleeding-specific breakdown was not separately reported at the same level of detail as its ischaemic components, the clearest evidence that this strategy's benefit is concentrated in bleeding rather than ischaemic protection comes from the pooled meta-analysis discussed below, where platelet-function-guided and genotype-guided approaches are analysed together as "guided" de-escalation [6].

Unguided de-escalation by switching: TOPIC and TALOS-AMI

TOPIC took a simpler approach: 645 stabilised ACS-PCI patients already tolerating prasugrel or ticagrelor were randomised at 1 month, without any platelet or genetic test, to either continue their potent inhibitor or switch outright to clopidogrel [2]. At 1 year, the composite of cardiovascular death, urgent revascularisation, stroke, or BARC grade 2 or higher bleeding occurred in 13.4% of the switched group versus 26.3% of the unchanged group (HR 0.48, 95% CI 0.34-0.68, p<0.01) [2]. This large effect was carried almost entirely by bleeding: BARC grade 2 or higher bleeding fell from 14.9% to 4.0% (HR 0.30, 0.18-0.50, p<0.01), while the trial reported no significant difference on ischaemic endpoints considered alone [2].

TALOS-AMI repeated this design at larger scale and with a formal non-inferiority framework: 2697 Korean patients stabilised after PCI for acute myocardial infarction were randomised at 1 month to unguided de-escalation from ticagrelor to clopidogrel or to continued ticagrelor [5]. The primary composite of cardiovascular death, myocardial infarction, stroke, or BARC grade 2, 3, or 5 bleeding at 12 months occurred in 4.6% of the de-escalated group versus 8.2% of the continued-ticagrelor group (HR 0.55, 95% CI 0.40-0.76) [5]. Bleeding fell from 5.6% to 3.0% (HR 0.52, 0.35-0.77), and the ischaemic component fell numerically from 3.1% to 2.1% (HR 0.69, 95% CI 0.42-1.14) -- a result the trial characterised as showing no significant increase in ischaemic events, but whose confidence interval does not exclude a true HR as high as 1.14, meaning a modest real increase in ischaemic risk with de-escalation cannot be statistically ruled out from this trial alone [5].

Unguided dose reduction: HOST-REDUCE-POLYTECH-ACS

HOST-REDUCE-POLYTECH-ACS tested a different lever on the same drug: rather than switching molecules, 2338 ACS-PCI patients on prasugrel were randomised at 1 month, again without any platelet or genetic test, to continue prasugrel 10 mg or step down to prasugrel 5 mg [3]. Net adverse clinical events at 1 year -- a composite of death, myocardial infarction, stent thrombosis, clinically driven revascularisation, stroke, and BARC grade 2 or higher bleeding -- occurred in 7.2% of the reduced-dose group versus 10.1% of the conventional-dose group (HR 0.70, 95% CI 0.52-0.92, p=0.012; number needed to treat 34) [3]. Figure 2 places this trial's bleeding-specific result on the same scale as TOPIC, TALOS-AMI, and the pooled meta-analysis estimates discussed below.

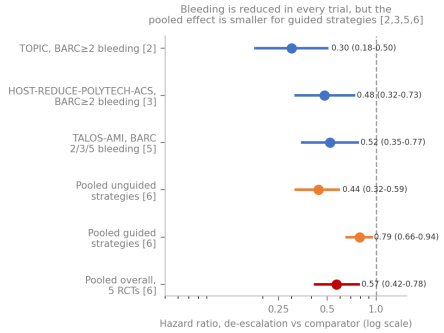

Figure 2: Bleeding hazard ratios (95% CI) for de-escalation versus comparator, individual trials and pooled meta-analysis estimates by strategy type [2,3,5,6].

As in TOPIC, the component-level picture in HOST-REDUCE-POLYTECH-ACS shows the benefit concentrated in bleeding: BARC grade 2 or higher bleeding fell from 5.9% to 2.9% (HR 0.48, 95% CI 0.32-0.73, p<0.001), while the ischaemic composite (death, myocardial infarction, stent thrombosis, stroke) moved from 1.8% to 1.4% (HR 0.76, 95% CI 0.40-1.45), a difference the trial did not consider statistically significant [3]. Table 2 sets these bleeding-versus-ischaemic component breakdowns out across the four trials that reported them separately.

Genotype-guided initial selection: POPular Genetics

POPular Genetics tested a fourth strategy that acts before any drug is even started: 2488 primary-PCI patients were randomised to either standard treatment with ticagrelor or prasugrel, or a CYP2C19 genotype-guided strategy in which carriers of loss-of-function alleles received a potent inhibitor while non-carriers received clopidogrel from the outset [4]. At 12 months, the combined thrombotic-and-bleeding primary endpoint occurred in 5.1% of the genotype-guided group versus 5.9% of the standard group, meeting non-inferiority (p<0.001) [4]. PLATO major-or-minor bleeding fell from 12.5% to 9.8% (p=0.04), while the purely ischaemic composite of death, myocardial infarction, stroke, or stent thrombosis moved from 3.3% to 2.7%, a difference not considered statistically significant [4]. Because genotyping identifies who can safely receive a lower-potency drug before treatment starts, rather than confirming adequate response after a trial period on a lower-potency drug, it shares TROPICAL-ACS's individualised, test-guided logic even though the two trials test different biomarkers at different times.

Pooling the evidence: is the ischaemic reassurance the same for every strategy?

A meta-analysis by Tavenier and colleagues pooled five randomised de-escalation trials (10,779 patients in total), grouping genotype-guided and platelet-function-guided strategies together as "guided" de-escalation and unguided switching and unguided dose reduction together as "unguided" de-escalation [6]. Across all five trials, de-escalation reduced BARC grade 2 or higher bleeding (HR 0.57, 95% CI 0.42-0.78) and reduced major adverse cardiac events (HR 0.77, 95% CI 0.62-0.96), with individual ischaemic components -- myocardial infarction (HR 0.75, 0.54-1.05), stent thrombosis (HR 0.68, 0.27-1.69), and stroke (HR 0.71, 0.43-1.18) -- each numerically favouring de-escalation without reaching significance on its own [6]. The honest complication in this otherwise reassuring pooled estimate is that the bleeding benefit itself was not uniform: unguided de-escalation produced a substantially larger pooled bleeding reduction (HR 0.44, 95% CI 0.32-0.59) than guided de-escalation (HR 0.79, 95% CI 0.66-0.94), and meta-regression found this difference statistically significant (interaction p=0.037) [6]. Figure 3 sets this divergence against the ischaemic outcome, for which the same guided-versus-unguided comparison showed no significant difference (interaction p=0.79) [6] -- meaning the open question this review set out to answer is really a question about the size of the bleeding benefit, not about a detected ischaemic cost, since no such cost has been statistically demonstrated for either family of strategies.

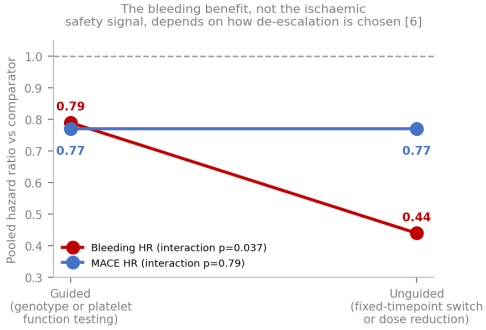

Figure 3: Pooled hazard ratios for guided versus unguided de-escalation strategies: bleeding benefit diverges by strategy type; ischaemic (MACE) benefit does not [6].

Figure 4 sets out how these four de-escalation strategies relate to one another as a taxonomy, and Figure 5 sets out the underlying pharmacological logic: potent inhibitors act with fixed intensity regardless of the individual patient, while clopidogrel's effect varies with CYP2C19 metaboliser status, which is exactly why two different testing strategies -- platelet function testing on treatment, and genotyping before treatment -- were each built to identify patients for whom a step down is safe.

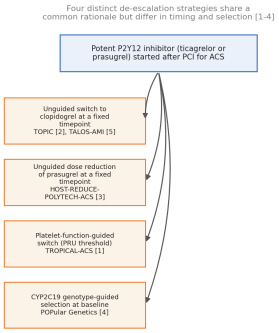

Figure 4: Taxonomy of the four de-escalation strategies reviewed here, sharing a common starting point but differing in timing and patient selection [1-4].

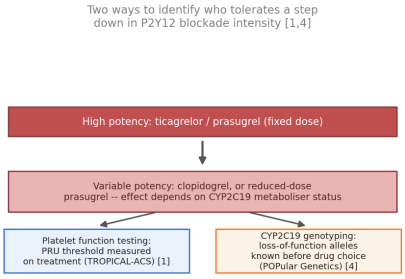

Figure 5: P2Y12 blockade intensity and the two testing strategies used to identify patients suitable for a step-down in potency [1,4].

POPular Genetics is the only strategy reviewed here that acts before the first dose is chosen, rather than after a period of observed treatment, which makes it operationally distinct from the other three even though its headline result -- a non-inferior combined endpoint alongside a significant bleeding reduction -- reads the same way [4].

Trial	N	Strategy	Comparator	Primary result at 1 year
TROPICAL-ACS [1]	2610	PFT-guided switch (day 14)	Standard prasugrel	7.3% vs 9.0% (HR 0.81, NI p=0.0004)
TOPIC [2]	645	Unguided switch (1 month)	Continued potent P2Y12	13.4% vs 26.3% (HR 0.48, p<0.01)
HOST-REDUCE- POLYTECH-ACS [3]	2338	Unguided dose reduction (1 month)	Prasugrel 10 mg	7.2% vs 10.1% (HR 0.70, p=0.012)
POPular Genetics [4]	2488	Genotype-guided selection (baseline)	Standard potent P2Y12	5.1% vs 5.9% (NI p<0.001)
TALOS-AMI [5]	2697	Unguided switch (1 month)	Continued ticagrelor	4.6% vs 8.2% (HR 0.55, 0.40-0.76)

Table 1: Design and headline primary-endpoint result of the five de-escalation trials reviewed here. Primary endpoint composition differs by trial [1,2,3,4,5].

Does the reassurance hold in higher-risk patients?

Two post hoc analyses ask whether the pattern above -- a real bleeding benefit alongside a numerically favourable but statistically inconclusive ischaemic signal -- still holds when attention narrows to the patients a clinician would most hesitate to de-escalate. A complex-PCI post hoc analysis of HOST-REDUCE-POLYTECH-ACS examined 705 patients meeting prespecified complexity criteria (349 randomised to reduced-dose, 356 to conventional-dose prasugrel): net adverse clinical events occurred in 8.2% of the de-escalated group versus 13.3% of the conventional-dose group (HR 0.60, 95% CI 0.37-0.95), a bleeding-driven benefit (BARC grade 2 or higher bleeding 1.8% versus 6.9%, HR 0.25, 0.10-0.61) that arrived without a significant rise in the separate MACE component (5.3% versus 6.0%, HR 0.88, 0.47-1.66) [7]. A high-atherothrombotic-risk post hoc analysis of TROPICAL-ACS, defining high risk as age 65 or older, polyvascular disease, or at least two of diabetes, smoking, and renal dysfunction, found the primary endpoint occurred in 10.5% of the guided de-escalation group versus 11.5% of the standard group among 990 high-risk patients (HR 0.90, 95% CI 0.61-1.32, p=0.586) and in 5.6% versus 7.7% among 1620 lower-risk patients (HR 0.71, 95% CI 0.48-1.04, p=0.082), with no significant interaction between risk stratum and treatment effect (p=0.394) [8]. Figure 6 sets these subgroup point estimates alongside the corresponding overall-trial results, and Table 3 sets the same figures out as text.

Interaction p=0.394 (TROPICAL-ACS risk strata) -- subgroup point estimates stay on the same side of null [1,3,7,8]

Population (N)	De-escalation	Comparator	HR (95% CI)
TROPICAL-ACS, overall (2610) [1]	7.3%	9.0%	0.81 (0.62-1.06)
TROPICAL-ACS, high ATR risk (990) [8]	10.5%	11.5%	0.90 (0.61-1.32)
TROPICAL-ACS, low ATR risk (1620) [8]	5.6%	7.7%	0.71 (0.48-1.04)
HOST-REDUCE-POLYTECH-ACS, overall (2338) [3]	7.2%	10.1%	0.70 (0.52-0.92)
HOST-REDUCE-POLYTECH-ACS, complex PCI (705) [7]	8.2%	13.3%	0.60 (0.37-0.95)

Figure 6: De-escalation point estimates by risk subgroup stay on the same side of the null in both TROPICAL-ACS atherothrombotic-risk strata and the HOST-REDUCE-POLYTECH-ACS complex-PCI subgroup [1,3,7,8].

Trial	Bleeding endpoint (de-esc vs comp.)	Ischaemic endpoint (de-esc vs comp.)
TOPIC [2]	BARC≥2: 4.0% vs 14.9% (HR 0.30, 0.18-0.50)	No significant difference (not separately quantified)
HOST-REDUCE- POLYTECH-ACS [3]	BARC≥2: 2.9% vs 5.9% (HR 0.48, 0.32-0.73)	1.4% vs 1.8% (HR 0.76, 0.40-1.45)
POPular Genetics [4]	PLATO maj./min.: 9.8% vs 12.5% (p=0.04)	2.7% vs 3.3% (not significant)
TALOS-AMI [5]	BARC 2/3/5: 3.0% vs 5.6% (HR 0.52, 0.35-0.77)	2.1% vs 3.1% (HR 0.69, 0.42-1.14)

Table 2: Bleeding and ischaemic component event rates, where separately reported, across four of the five trials [2,3,4,5].

Discussion

Taken as a set, these five trials, one pooling meta-analysis, and two risk-stratified subgroup analyses support a real and clinically meaningful bleeding reduction from antiplatelet de-escalation after PCI for ACS, achieved through four mechanistically distinct routes, none of which has produced a statistically demonstrated rise in ischaemic events. That is a genuinely reassuring body of evidence, and it is also, on close reading, a body of evidence that reassures more confidently about some strategies than about others. The meta-analysis's central honest finding is not that de-escalation is unsafe -- pooled MACE favours de-escalation (HR 0.77) -- but that unguided strategies deliver roughly twice the pooled bleeding-risk reduction of guided strategies (HR 0.44 versus 0.79, interaction p=0.037), a difference too large and too statistically robust to describe as noise [6]. Individual trials, meanwhile, are consistently underpowered to exclude a real ischaemic cost on their own terms: TALOS-AMI's ischaemic hazard ratio of 0.69 carries a confidence interval reaching 1.14, and HOST-REDUCE-POLYTECH-ACS's reaches 1.45, so "no significant increase" in these trials describes an absence of proof rather than proof of absence [3,5].

For the practising interventional cardiologist, this leaves a genuinely two-track picture rather than a single verdict. Unguided strategies -- switching to clopidogrel or halving the prasugrel dose at a fixed timepoint, typically 1 month after PCI -- have the largest, most consistent bleeding benefit in this literature and require no additional test, but they are applied uniformly to a population that includes some patients who might still benefit from full-intensity inhibition [2,3,5]. Guided strategies -- platelet-function testing or CYP2C19 genotyping -- individualise that choice and produced a smaller but still real bleeding benefit in the pooled analysis, and the two dedicated subgroup analyses reviewed here are specifically reassuring for the guided, individualised logic: neither TROPICAL-ACS's high-atherothrombotic-risk stratum nor HOST-REDUCE-POLYTECH-ACS's complex-PCI stratum showed a significant interaction suggesting the strategy stops working, or starts causing harm, in exactly the patients where a clinician would most want reassurance before de-escalating [7,8]. The 2020 ESC guideline for non-ST-elevation ACS reflects this same balance of confidence, recommending consideration of platelet-function or genotype-guided de-escalation as an alternative DAPT strategy, a conditional rather than unconditional endorsement that matches the trial evidence's own conditional certainty [9].

A practical reading of the taxonomy in Figure 4 is that the choice between these four strategies is currently less a question of which one is safe -- all four look safe on the ischaemic side within the limits of the evidence -- and more a question of what infrastructure a given centre already has. A centre without same-day platelet function testing or point-of-care CYP2C19 genotyping can still offer the unguided switch or dose-reduction strategies, accepting one that the pooled meta-analysis suggests may in fact deliver a larger average bleeding benefit, without tailoring the decision to any single patient's measured biology [1,2,3,4,5,6].

Study limitations

This is a narrative review rather than a systematic review with a pre-registered protocol and a fixed search strategy, and its conclusions rest on five trials, one meta-analysis, and two subgroup analyses rather than an exhaustive search of the de-escalation literature. The five trials differ in more than just guided-versus-unguided design: they used different P2Y12 inhibitors as the starting point (prasugrel in TROPICAL-ACS and HOST-REDUCE-POLYTECH-ACS, a mix of prasugrel and ticagrelor in TOPIC, ticagrelor in TALOS-AMI, either drug in POPular Genetics), different de-escalation timepoints (14 days in TROPICAL-ACS, 1 month in the three unguided trials, baseline in POPular Genetics), and different primary endpoint definitions, so the review's summary that "bleeding falls, ischaemic events do not significantly rise" is a pattern observed across a heterogeneous set of interventions rather than a single reproducible number a clinician can quote for every de-escalation decision [1,2,3,4,5].

A further point worth stating plainly for practice: none of the five headline trials, and neither of the two subgroup analyses reviewed here, was individually powered to demonstrate ischaemic non-inferiority with the precision that the bleeding endpoint achieved -- ischaemic event counts were small enough in every trial that confidence intervals on the ischaemic hazard ratio remained wide and, in TALOS-AMI and HOST-REDUCE-POLYTECH-ACS, extended above 1 [3,5]. The pooled meta-analysis narrows those intervals considerably and its overall MACE estimate (HR 0.77, 95% CI 0.62-0.96) does exclude a large ischaemic penalty, but a meta-analysis inherits the specific mix of strategies and populations its five constituent trials happened to test, predominantly East Asian populations for the unguided dose- and switch-based trials and predominantly European populations for the guided trials, and cannot fully substitute for a single trial powered on ischaemic outcomes as its primary hypothesis [6]. Follow-up beyond 12 months is limited across this literature, so durability of both the bleeding benefit and the ischaemic reassurance beyond the first year after PCI remains an open question rather than an established fact. Finally, the two subgroup analyses reviewed here, while reassuring on their point estimates and interaction tests, are themselves post hoc rather than prespecified primary analyses, and their own confidence intervals -- particularly TROPICAL-ACS's high-risk stratum, spanning 0.61 to 1.32 -- are wide enough that a true difference by risk category cannot be fully excluded either [7,8].

Finally, this review grouped TROPICAL-ACS and POPular Genetics together as "guided" strategies because both individualise treatment with a biological measurement, but they measure different things at different times -- on-treatment platelet reactivity at day 14 in one case, a fixed genotype known before the first dose in the other -- and a reader should not assume the two are interchangeable simply because the meta-analysis pooled them for its guided-versus-unguided comparison [1,4,6].

Conclusion

Across four distinct routes -- unguided switching, unguided dose reduction, platelet-function-guided switching, and genotype-guided initial selection -- antiplatelet de-escalation after PCI for ACS reduces bleeding, in most trials substantially, without a statistically demonstrated rise in ischaemic events, including in complex-PCI and high-atherothrombotic-risk subgroups specifically examined for this concern. The honest complication in this literature is not a detected ischaemic cost but an unresolved question about how large the bleeding benefit really is for guided, individualised strategies compared with unguided ones, and an acknowledgement that no individual trial reviewed here was powered to convert "no significant ischaemic increase" into the stronger claim of proven ischaemic equivalence. For now, de-escalation belongs in practice as a considered, evidence-supported step down rather than either a blanket default or an unproven risk, with the choice between guided and unguided approaches resting on local access to platelet or genetic testing as much as on any difference in demonstrated safety.

Analysis	N	De-escalation	Comparator	HR (95% CI)
TROPICAL-ACS, overall [1]	2610	7.3%	9.0%	0.81 (0.62-1.06)
TROPICAL-ACS, high ATR risk [8]	990	10.5%	11.5%	0.90 (0.61-1.32)
TROPICAL-ACS, low ATR risk [8]	1620	5.6%	7.7%	0.71 (0.48-1.04)
HOST-REDUCE-POLYTECH-ACS, overall [3]	2338	7.2%	10.1%	0.70 (0.52-0.92)
HOST-REDUCE-POLYTECH-ACS, complex PCI [7]	705	8.2%	13.3%	0.60 (0.37-0.95)

Table 3: Primary-endpoint point estimates by risk subgroup, alongside each trial's overall result, for the two post hoc analyses reviewed [1,3,7,8].

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