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Antimicrobial Catheter Technologies: From Chemistry and Manufacturing to *In Vivo* Performance

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Introduction

Antimicrobial (AM) performance claims for catheters vary across manufacturers, as they are often based on different bench-top and animal test methods. These differences can make direct product comparisons misleading. Appropriate catheter selection requires evaluating whether the test methods are comparable to simulate real-world clinical conditions, as well as understanding the mechanisms of action of catheter's AM technology, and its limitations.

Methods

- Classified vascular catheters by AM technology, it's mechanism of action and limitations, as well as manufacturing process based on publicly available manufacturer's information and scientific data.
- Performed simultaneous testing of select technologies to compare AM efficacy, hemocompatibility, and antithrombogenic (AT) performance.
- Utilized *in vitro* and *in vivo* models for testing - that were designed to simulate clinically relevant use conditions.

Limitations & Disclosure

- *In vitro*/*in vivo* sheep model results may not reflect clinical outcomes.
- Chlorhexidine-based catheters are manufactured by Teleflex.
- Author is a Teleflex employee.
- Study funded by Teleflex; data on file.

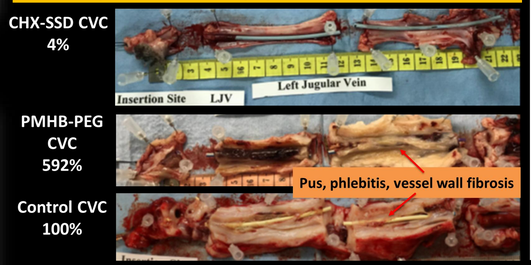
Results

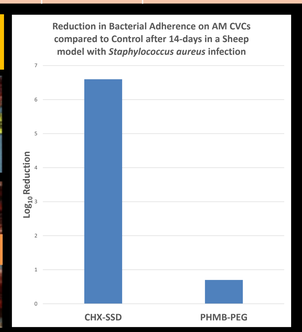
Category	Technology	Technology Application Process	Mechanism of Action	Antimicrobial Effect	Antithrombogenic /Antibiofouling Effect	Limitations
Eluting (Active Release)	Minocycline/ Rifampicin	Surface impregnation	Antibiotic release; inhibits protein synthesis and RNA transcription respectively	✓	✗	Resistance risk, limited protection against gram-negative bacteria and none against fungi
	Rifampicin + Miconazole	Surface impregnation	Antibiotic/antifungal release; targets RNA transcription and membrane-associated enzyme respectively	✓	✗	Resistance risk, limited gram-negative protection
	Ionic Silver	Surface impregnation or bulk distribution depending on manufacturer	Silver ion release; multi-target binding to proteins, membranes, and DNA with oxidative stress–mediated damage Silver ion release; multi-target binding to proteins, membranes, and DNA with oxidative stress–mediated damage	✓	✗	Rapid inactivation in protein-rich environments due to silver ion binding to plasma proteins, chloride ions, and sulfur-containing biomolecules Rapid inactivation in protein-rich environments due to silver ion binding to plasma proteins, chloride ions, and sulfur-containing biomolecules
	Silver/Platinum/Carbon	Bulk incorporation		✓	✗	
	Chlorhexidine	Surface impregnation	Chlorhexidine cation release; Membrane disruption, destabilization and cell lysis via cationic binding	✓	✓	Requires controlled kinetics to avoid early burst depletion; potential insertion site bleeding at initial higher dose release
	Chlorhexidine + Silver sulfadiazine	Surface impregnation	Dual antiseptic release; chlorhexidine cation causes membrane rupture and silver, and sulfadiazine ions bind to proteins, membranes, and DNA with oxidative stress–mediated damage and folate-pathway inhibition	✓	✗	Limited activity in long dwell times
Non-eluting (Passive Inhibition)	PHMB + PEG	Covalent bonding	Contact kill by PHMB through membrane disruption, destabilization and cell lysis and PEG reduces microbial and protein adhesion by forming a hydrated, sterically repulsive surface layer	✓	✓	Limited antimicrobial activity as kill depends direct microbial contact; short-term resistance to biofouling
	Gold/Silver/ Palladium alloy	Bulk incorporation	Reduces microbial attachment via electrochemical effect altering surface redox potential	✓	✗	Limited antimicrobial activity as kill depends direct microbial contact
	Hydrogel	Bulk incorporation or surface coating depending on manufacturer	Hydrophilic network absorbs water to form hydrated surface layer reducing protein layer deposition through steric repulsion, which leads to indirect thrombus reduction	✗	✓	No kill mechanism, ineffective if contamination occurs; does not suppress planktonic load
	Poly(2-methoxyethylacrylate) (PMEA)	Surface coating	Amphiphilic; creates intermediate water layer reducing protein layer deposition which leads to indirect thrombus reduction	✗	✓	
	Fluoropolymer / Fluoro-oligomer	Bulk incorporation	Creates low–surface energy surface reducing protein layer deposition which leads to indirect thrombus reduction	✗	✓	

Organism	% Reduction in Microbial Colonization after 24-hours compared to initial challenge		
	Plain PICC	Fluoro-Polymer PICC	Chlorhexidine PICC
Staphylococcus epidermidis ATCC 35983	-1,499.22	-1,113.13	100
Candida albicans ATCC 10231	82.96	98.55	100
Enterococcus faecium ATCC 35667	-249.87	-136.54	100
Enterococcus faecalis ATCC 49532	-3,648.94	-5,195.52	100
Acinetobacter baumannii ATCC 19606	-57,964.24	-63,566.17	100
Escherichia coli ATCC 35218	-10,465.95	-6,880.86	100
Pseudomonas aeruginosa ATCC 27853	-26,440.48	-52,855.22	100
Klebsiella pneumoniae ATCC 10031	-163,547.26	-72,998.55	100
Citrobacter freundii ATCC 8090	-10,465.95	-15,892.22	100
Serratia marcescens ATCC 8100	-20,033.01	-37,389.42	100
Methicillin Resistant *Staphylococcus aureus* ATCC 33591	-1,499.22	-2,057.29	100
Methicillin Resistant *Staphylococcus epidermidis* ATCC 51625	97.35	99.30	100
Vancomycin Resistant *Enterococcus faecalis* ATCC 51299	-2,376.90	-1,653.51	100
Carbapenem Resistant *Enterobacter cloacae* BAA-2468	-21,472.91	-15,892.22	100
Carbapenem Resistant *Escherichia coli* BAA-2469	-219.09	-701.51	100
Carbapenem Resistant *Klebsiella pneumoniae* BAA-2470	-42,943.62	-11,755.20	100

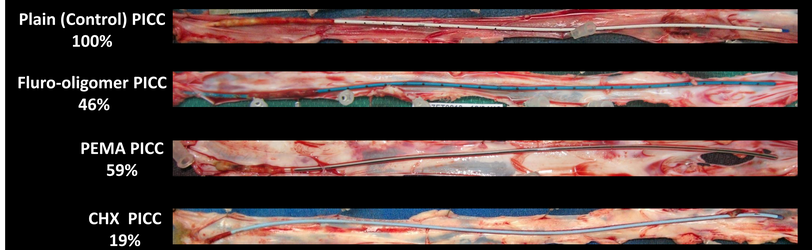
Organism	% Reduction in Microbial Colonization after 7-days compared to Initial Challenge		
	Minocycline/Rifampin CVC	Silver/Carbon/Platinum CVC	Chlorhexidine/Silver sulfadiazine CVC
Methicillin Susceptible *Staphylococcus aureus* ATCC 29213	100	-140	100
Staphylococcus epidermidis ATCC 35983	100	99.96	100
Candida albicans ATCC 10231	-200	95	100
Enterococcus faecium ATCC 35667	99.96	99.96	100
Enterococcus faecalis ATCC 49532	99.96	-380	100
Acinetobacter baumannii ATCC 19606	100	-860	99.76
Acinetobacter calcoaceticus CMPC-1	100	-32	100
Escherichia coli ATCC 35218	100	60	100
Pseudomonas aeruginosa ATCC 27853	64	98.64	100
Klebsiella pneumoniae ATCC 10031	100	99.92	100
Enterobacter cloacae ATCC 13047	100	-440	100
Citrobacter freundii ATCC 8090	100	-100	100
Serratia marcescens ATCC 8100	100	99.98	100
Methicillin Resistant *Staphylococcus aureus* ATCC 33591	100	20	100
Carbapenem Resistant *Enterobacter cloacae* BAA-2468	80	-40	99.96
Carbapenem Resistant *Escherichia coli* BAA-2469	99.84	98.60	100
Carbapenem Resistant *Escherichia coli* BAA-2471	99.86	94.40	100
Carbapenem Resistant *Klebsiella pneumoniae* BAA-2470	40	-200	100
Carbapenem Resistant *Klebsiella pneumoniae* BAA-2472	74	86	99.98
Carbapenem Resistant *Klebsiella pneumoniae* BAA-2473	-40	20	100

% Thrombus Weight compared to Control after 14-days in a sheep model with *Staphylococcus aureus* infection





% Thrombus Weight Compared to Control after 30-days in a sheep model



Conclusions

- Chlorhexidine-eluting PICCs and CVCs demonstrated the broadest spectrum and greatest magnitude of antimicrobial activity among all antimicrobial catheters evaluated.
- Chlorhexidine-eluting catheters reduced microbial colonization and thrombus accumulation more effectively than non-eluting fluoropolymer- and PHMB+PEG-based catheters.
- In a 2-hour *in vitro* blood flow loop model, platelet adhesion was comparable among non-eluting PICCs (data not shown).
- In clinically simulated ovine models, chlorhexidine-eluting catheters led to significantly higher thrombus reduction compared to non-eluting fluoropolymer- and PHMB+PEG-based catheters.

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