RESEARCH PAPER

An Integrated In-Vitro Platform for Chemical Renal Denervation: Neuronal Screening, Vincristine Quantification, and Controlled Release from PLGA-Coated Stents

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Abstract

Background: Renal sympathetic signalling contributes to blood-pressure regulation, and renal denervation is an adjunctive option for selected patients whose hypertension remains uncontrolled despite optimized treatment. This study investigated an alternative chemical-denervation concept in which a neuroactive compound is delivered locally from a biodegradable, drug-eluting renal stent. Methods: Thirteen neuroactive or neurotoxic compounds were screened in rat dorsal root ganglion neurones (RDRGN) using an MTT assay. Vincristine-associated changes in organelle transport were examined by fluorescence imaging and kymograph analysis. A liquid chromatography–tandem mass spectrometry (LC–MS/MS) assay was developed for vincristine quantification. Vincristine–poly(lactic-co-glycolic acid) (PLGA) coatings containing PLGA 20:80, 50:50, or 75:25 were applied to cobalt–chromium stents and assessed by gravimetry, scanning electron microscopy, Fourier-transform infrared spectroscopy, drug-content analysis, and dynamic in-vitro release testing using USP Apparatus IV. Results: Vincristine had the lowest MTT-derived IC_{50} in the screening panel (1.24 µg), followed by carboplatin (1.35 µg), cisplatin (1.46 µg), and vinblastine (1.56 µg). Vincristine exposure was associated with slower anterograde and retrograde transport, shorter run lengths, more pausing, and a reduced mobile fraction. The LC–MS/MS method used the MRM transition m/z 825.3→807.3, showed linearity from 10–150 ppm (R^2=0.995734), and had reported limits of detection and quantification of 3 and 10 ppm, respectively. Coated stents contained approximately 124–125 µg vincristine per device. Mean cumulative release from PLGA 20:80 was 27.36% by day 3 and 98.19% by day 30; PLGA 50:50 released 11.17% by day 3 and 96.14% by day 45; PLGA 75:25 released 8.66% by day 3 and approximately 98.27% by day 90. Conclusion: The combined findings establish in-vitro feasibility for screening a candidate neurolytic compound, quantifying it, and controlling its release from PLGA-coated stents. They do not establish selective renal sympathetic denervation, vascular safety, blood-pressure efficacy, or clinical benefit. These questions require staged in-vivo evaluation before therapeutic claims can be made.

Keywords: resistant hypertension; renal denervation; vincristine; PLGA; drug-eluting stent; LC–MS/MS; axonal transport; in-vitro release

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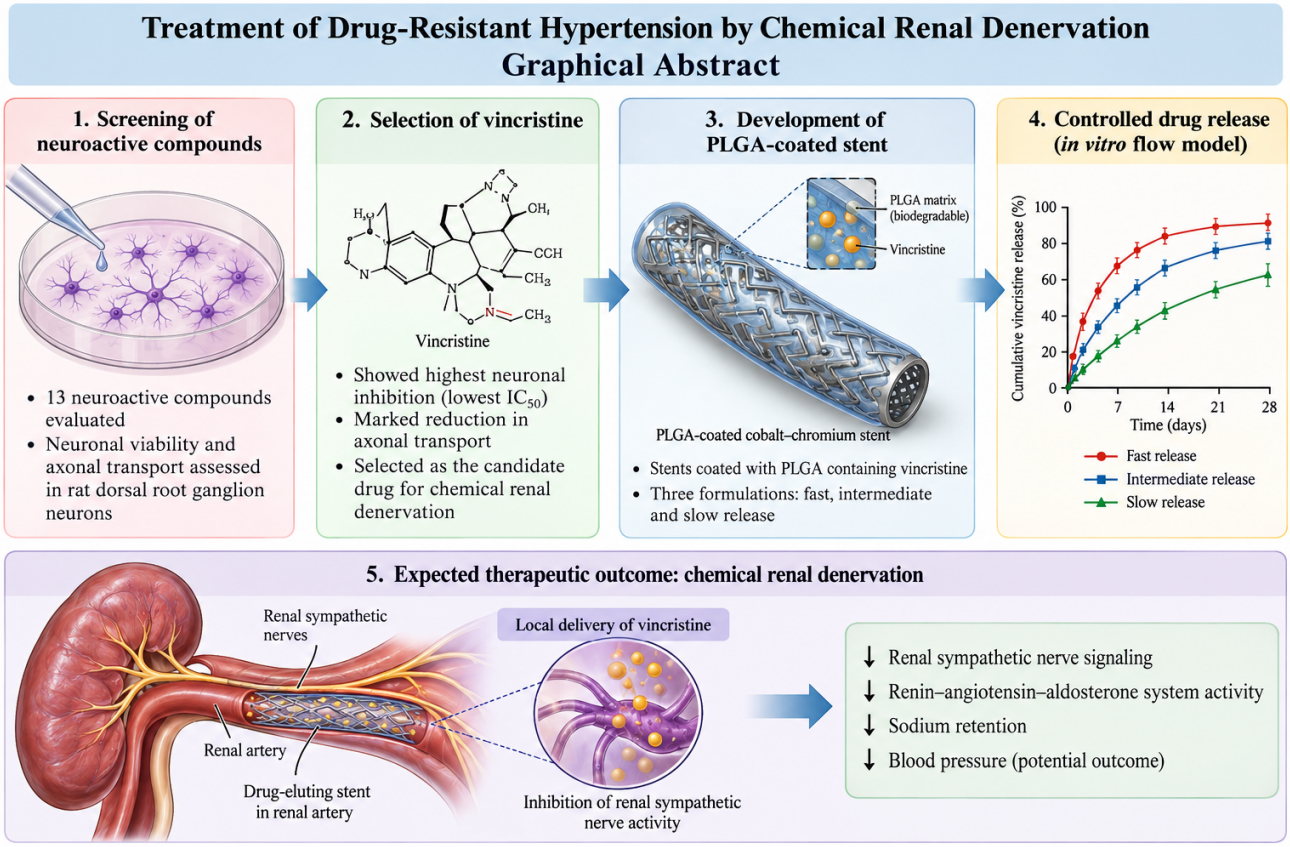

1. Introduction

Resistant hypertension is generally defined as blood pressure that remains above target despite treatment with three complementary antihypertensive drug classes at maximally tolerated doses, commonly including a renin–angiotensin system blocker, a long-acting calcium-channel blocker, and a diuretic. Before resistance is confirmed, measurement error, poor adherence, white-coat effects, inadequate dosing, and secondary causes should be assessed. Persistent uncontrolled pressure is associated with an increased burden of cardiovascular and renal complications [1,2].

The kidney is both a target and a regulator of sympathetic activity. Renal sympathetic nerves influence renal vascular tone, tubular sodium reabsorption, and renin release; these effects interact with the renin–angiotensin–aldosterone system and contribute to blood-pressure homeostasis. Catheter-based renal denervation seeks to interrupt renal sympathetic signalling. Radiofrequency and ultrasound approaches have been evaluated clinically, but responses vary between patients and trials, and these procedures require specialized catheter systems and careful patient selection [3,4].

Chemical renal denervation offers a different concept: a neurolytic compound is delivered close to periarterial sympathetic fibres rather than relying exclusively on thermal energy transmitted from within the arterial lumen. The approach presents a key formulation challenge. Local exposure must be sufficient and durable to affect the intended nerves while avoiding injury to the arterial wall, adjacent tissues, or systemic organs. A controlled-release device could allow the duration of local delivery to be varied and studied systematically.

Vincristine disrupts microtubule dynamics and can impair axonal transport, a process required for long-distance movement of organelles and cellular materials in neurons. Its neurotoxicity makes it a candidate for investigation as a local neurolytic agent, but that same property raises important safety questions and does not imply selectivity for renal sympathetic nerves. The present work therefore combined comparative neuronal screening, functional examination of axonal transport, analytical method development, and stent formulation. The objective was to assess the in-vitro feasibility of a vincristine-eluting PLGA-coated renal stent and to determine whether PLGA composition could produce distinct release durations.

2. Materials and Methods

2.1. Materials and study design

Thirteen compounds were screened: vincristine, vinblastine, paclitaxel, docetaxel, cisplatin, oxaliplatin, carboplatin, bortezomib, digoxin, strophanthidin, digitoxin, proscillaridin, and ouabain. Vincristine sulphate and PLGA grades with lactide:glycolide ratios of 20:80, 50:50, and 75:25 were used for formulation. Cobalt–chromium stents were coated with vincristine–PLGA mixtures. The overall workflow comprised neuronal viability screening, axonal-transport imaging,

LC–MS/MS method validation, coating characterization, drug-content determination, and in-vitro release testing.

2.2. MTT cytotoxicity screening

RDRGN cells obtained from the National Centre for Cell Sciences, Pune, were subcultured at ClinTech Solutions, Hyderabad. Cells were seeded in triplicate in 96-well plates at 100 µL per well and equilibrated overnight in drug-free DMEM. Test compounds were added over the relevant concentration ranges and incubated for 24 h at 37 °C in humidified 5% CO_2. MTT solution (1 mg/mL) was then added for 4 h. After removal of medium, formazan was dissolved in 150 µL DMSO and absorbance was measured at 540 nm. Percentage inhibition was calculated relative to the mean untreated control, and IC_{50} values were derived from concentration–response relationships. A lower IC_{50} was interpreted as greater cytotoxic potency in this assay; it was not treated as evidence of sympathetic-neuron selectivity.

2.3. Axonal-transport imaging

RDRGN cultures were maintained in high-glucose DMEM containing 10% horse serum, 5% fetal bovine serum, and 1% penicillin–streptomycin at 37 °C and 5% CO_2. Differentiation was induced with nerve growth factor (50 ng/mL) for 5–7 days. Differentiated cells were transferred to poly-L-lysine-coated glass-bottom dishes and exposed to selected vincristine concentrations for 24–48 h. Lysosomes were labelled with LysoTracker Red DND-99 (50 nM) and mitochondria with MitoTracker Green FM (100 nM). Live-cell sequences were recorded using an inverted fluorescence microscope with a CCD camera and environmental control at 1–5 frames/s for 5–10 min. ImageJ and kymograph analysis were used to assess anterograde and retrograde velocity, mobile fraction, run length, and transport frequency.

2.4. LC–MS/MS method development and validation

Vincristine was analyzed using a Waters Acquity UPLC system coupled to a Quattro Premier XF triple-quadrupole mass spectrometer. Positive electrospray ionization and multiple reaction monitoring were used with the transition m/z 825.3→807.3. Chromatographic separation used a Thermo Hypurity C18 column (50 × 4.6 mm, 5 µm). The mobile phase comprised 2 mM ammonium formate with 0.2% formic acid and acetonitrile (30:70, v/v), at 0.5 mL/min; injection volume was 10 µL. The retention time was approximately 1.21 min and the total run time 2.5 min. Calibration standards were prepared in methanol at 10, 25, 50, 75, 100, 125, and 150 ppm. Validation assessed system suitability, carryover, linearity, LOD, LOQ, accuracy, intra-day precision, inter-day precision, stability, and matrix interference.

2.5. Stent coating and characterization

Vincristine and PLGA were combined at a 1:1 weight ratio. The target loading was approximately 124 µg vincristine and 124 µg PLGA per stent, corresponding to a total coating mass of about 248 µg. Coating solutions were prepared at 1 mg/mL and applied to cleaned cobalt–chromium stents by controlled spray coating; coated devices were dried under nitrogen. Twenty-seven stents were prepared, with nine allocated to each PLGA composition. For each composition, three stents were used for drug-content analysis and six for release testing. Coating mass was determined gravimetrically from pre- and post-coating weights. Scanning electron microscopy was used to inspect coating morphology. FTIR spectra of pure vincristine and the three drug–polymer formulations were compared over 4000–400 cm^{-1} for characteristic bands and evidence of potential interactions.

2.6. Drug-content analysis and in-vitro release

Each coated stent was immersed in 5 mL acetonitrile and sonicated for 30 min at room temperature. Extracts were centrifuged at 3,000 rpm for 10 min and the supernatant was analyzed by the validated LC–MS/MS method. In-vitro release was evaluated using USP Apparatus IV (Sotax CE7) in a closed-loop configuration with PBS (pH 7.4) maintained at 37 ± 0.5 °C and a flow rate of 8 mL/min. Six stents per formulation were tested. Samples were collected on days 0, 3, 7, 15, 21, and 30 for PLGA 20:80; days 0, 3, 7, 15, 30, and 45 for PLGA 50:50; and days 0, 3, 7, 15, 30, 60, and 90 for PLGA 75:25. Samples were filtered through 0.22-µm membranes, diluted when needed, stored at 4 °C, and quantified by LC–MS/MS. Cumulative amounts and percentages released were calculated from measured concentrations.

2.7. Data handling

Results are reported as means and standard deviations. The release experiments used six stents per formulation. The source material does not consistently report inferential statistical testing or model-based release-kinetic fitting; therefore, the manuscript describes observed profiles comparatively and does not claim statistically significant between-formulation differences or a formal kinetic model.

3. Results

3.1. Neuronal screening identified vincristine as the most potent candidate

Vincristine produced the lowest reported IC_{50} in the RDRGN MTT assay (1.24 µg). The next lowest values were carboplatin (1.35 µg), cisplatin (1.46 µg), and vinblastine (1.56 µg). Other reported IC_{50} values were oxaliplatin, 5.64 µg; bortezomib, 4.62 µg; digitoxin, 12.91 µg; proscillaridin, 18.94 µg; docetaxel, 19.28 µg; strophanthidin, 20.14 µg; ouabain, 20.84 µg; paclitaxel, 24.56 µg; and digoxin, 34.25 µg. The range of values indicated substantial differences in cytotoxic potency under the assay conditions.

Table 1. MTT-derived IC_{50} values

Compound	IC_{50} (µg)
Vincristine	1.24
Carboplatin	1.35
Cisplatin	1.46

Vinblastine	1.56
Bortezomib	4.62
Oxaliplatin	5.64
Digitoxin	12.91
Proscillaridin	18.94
Docetaxel	19.28
Strophanthidin	20.14
Ouabain	20.84
Paclitaxel	24.56
Digoxin	34.25

3.2. Vincristine altered axonal transport

Kymograph analysis showed movement of labelled organelles in both anterograde and retrograde directions in neuronal processes. Following vincristine exposure, trajectories were described as slower and shorter, with more pauses and a smaller mobile fraction than in untreated cells. These observations are consistent with impaired microtubule-dependent transport.

3.3. LC–MS/MS assay performance

The assay used a short chromatographic run, with vincristine eluting at approximately 1.21 min. Calibration was linear over 10–150 ppm, with the reported regression equation $y = 698.983x - 2606.21$ and $R^2 = 0.995734$. The reported LOD and LOQ were 3 and 10 ppm. System-suitability testing across six injections at 100 ppm gave a peak-area %RSD of 2.27% and retention-time %RSD of 0.45%. Reported recovery across LOQ, 50%, 100%, and 150% levels was within 91–106%, while intra-day and inter-day precision ranged from 1.2% to 3.1%. The method was subsequently applied to stent extracts and release samples.

3.4. Coating and drug loading

Vincristine was incorporated into all three PLGA coating compositions. Gravimetric analysis and LC–MS/MS content measurements indicated coating loads near the target and drug content of approximately 124–125 µg per stent, with low reported variability across groups. SEM images were described as showing adherent coatings without major visible discontinuities. FTIR comparison was used to examine drug–polymer compatibility; these data support formulation characterization but do not alone establish long-term chemical stability or in-vivo compatibility.

3.5. PLGA composition produced distinct release durations

Table 2 compares selected cumulative release measurements for the three PLGA compositions and highlights their different release durations. The PLGA 20:80 formulation showed the greatest early release, reaching 27.36 ± 1.79% by day 3, 50.01 ± 0.83% by day 7, 71.06 ± 1.99% by day 15, 88.32 ± 2.54% by day 21, and 98.19 ± 0.46% by day 30. The PLGA 50:50 formulation released 11.17 ± 0.25% by day 3, 21.48 ± 0.39% by day 7, 51.26 ± 2.24% by day 15, 84.77 ± 1.19% by day 30, and 96.14 ± 2.97% by day 45. The PLGA 75:25 formulation had the lowest day-3 release (8.66 ± 0.23%) and reached 51.78 ± 2.36% by day 7, 62.24 ± 2.07% by day 15, 71.80% by day 30, 94.80 ± 2.33% by day 60, and 98.27 ± 2.55% by day 90. Taken together, the data in Table 2 indicate that changing the PLGA ratio altered both the early release fraction and the time required to approach complete release.

Table 2. Selected cumulative release results (mean ± SD)

PLGA ratio	Day 3 release (%)	Near-terminal release (%)	Study endpoint
20:80	27.36 ± 1.79	98.19 ± 0.46 (day 30)	30 days
50:50	11.17 ± 0.25	96.14 ± 2.97 (day 45)	45 days
75:25	8.66 ± 0.23	98.27 ± 2.55 (day 90)	90 days

Note: Release endpoints differ by formulation, so terminal values should be interpreted with the stated observation period.

The profiles were reproducible across six stents per formulation, with generally low standard deviations at reported time points. PLGA 20:80 showed the greatest early release and reached near-complete release within 30 days. PLGA 50:50 showed a smaller initial fraction and near-complete release by day 45. PLGA 75:25 had the lowest reported day-3 release and continued release through day 90. These data demonstrate that changing PLGA composition altered the timing of vincristine release under the selected in-vitro conditions.

4. Discussion

This study links candidate selection, analytical measurement, and device formulation in a single in-vitro development sequence. The lowest MTT-derived IC_{50} was observed with vincristine, and the accompanying kymograph findings showed disruption of bidirectional organelle transport. These two observations provide a rationale for further investigation of vincristine as a local neurolytic candidate. However, MTT is a broad measure of metabolic activity and viability, and dorsal root ganglion neurones are not a direct model of renal sympathetic fibres. The results therefore demonstrate neuronal toxicity and transport impairment in the chosen model, not selective renal denervation.

The LC–MS/MS assay was important to the formulation work because estimates of loading and cumulative release depend on reliable drug quantification. The reported calibration, sensitivity, precision, and recovery support its use for the in-vitro samples studied. Transfer to plasma, renal arterial tissue, or periarterial tissue

would require separate matrix-specific bioanalytical validation, including recovery, matrix effects, lower quantification limits, stability, and an appropriate internal standard where necessary.

The release profiles summarized in Table 2 show that the three PLGA compositions produced distinguishable release patterns. PLGA 20:80 showed a relatively large early release fraction and reached approximately 98% release by day 30. PLGA 50:50 had a more moderate early fraction and approached 96% release by day 45, whereas PLGA 75:25 extended release to approximately 90 days. These data support the use of polymer composition as a formulation variable for modifying in-vitro release duration; they do not, by themselves, establish the duration of drug exposure at periarterial nerves.

On release behaviour alone, PLGA 50:50 is a reasonable intermediate formulation to carry forward for comparative preclinical testing because it combines a moderate early fraction with delivery over several weeks. This is a provisional formulation choice rather than a claim of therapeutic superiority. The optimal profile will depend on how much drug reaches periarterial nerves, how long that exposure persists, whether nerve injury is durable, and whether the vessel wall and adjacent structures remain safe. An in-vitro release curve cannot answer these questions.

Several limitations should be considered. First, the study was predominantly in vitro; no animal implantation, renal-nerve histology, tissue pharmacokinetics, systemic exposure assessment, blood-pressure measurement, or clinical evaluation was reported. Second, the screening endpoint does not establish selectivity for sympathetic neurons. Third, the concentration units and assay-level details for the reported IC_{50} values should be checked against original records, and the complete numerical transport data should be added if available. Fourth, the release studies used different observation windows for the three formulations, which should be stated clearly when comparing endpoints. Finally, local delivery does not eliminate systemic exposure risk, and vincristine's neurotoxicity makes evaluation of off-target effects essential.

Future work should proceed through a staged preclinical program: (i) confirm coating reproducibility and drug stability; (ii) assess endothelial-cell compatibility, thrombogenicity, vascular inflammation, and vessel healing; (iii) quantify vincristine in the arterial wall, periarterial tissue, plasma, and relevant organs after implantation; (iv) examine renal sympathetic nerve structure and function; and (v) measure renal function and blood-pressure outcomes with appropriate controls. A formal in-vitro–in-vivo correlation should only be developed after paired release, tissue-exposure, and pharmacodynamic data are available.

5. Conclusion

The integrated findings support the in-vitro feasibility of a vincristine-eluting PLGA-coated stent as a platform for investigating chemical renal denervation. Vincristine showed the lowest IC_{50} among the 13 compounds screened and was associated with impaired axonal transport. A short-run LC–MS/MS method enabled quantification of vincristine in stent extracts and release samples. Coated stents carried approximately 124–125 µg vincristine per device, while PLGA 20:80, 50:50, and 75:25 generated fast, intermediate, and prolonged release profiles, respectively. The data do not establish in-vivo denervation, vascular safety, blood-pressure reduction, or clinical efficacy. The next step is a controlled preclinical assessment of local exposure, neural effects, vascular compatibility, systemic exposure, and haemodynamic response.

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