

THE SYNTHESIS AND CHARACTERIZATION OF NOVEL PLATINUM(II)
COMPLEXES CONTAINING BULKY AROMATIC GROUPS

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Abstract

Six iminopyridine ligands were prepared from the condensation of 6-methyl-2-pyridinecarboxyaldehyde and the corresponding primary amines containing bulky cycloalkyl groups. Addition of these ligands to $[\text{PtCl}_2(\text{coe})]_2$ (coe = *cis*-cyclooctene) gave the desired platinum(II) coordination complexes in moderate to high yields. All compounds were fully characterized using FT-IR and multinuclear NMR spectroscopy. Solid compounds were subjected to melting point analysis. Elemental analysis (EA) was performed on three of the platinum(II) complexes. Future work will involve completing elemental analysis studies and performing biological testing. X-ray diffraction studies must be performed to support the existing characterization data.

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List of Abbreviations

δ	chemical shift
Å	Angstrom
$^{\circ}\text{C}$	degrees Celsius
$^{13}\text{C}\{^1\text{H}\}$	proton-decoupled ^{13}C
Ar	aryl (spectra)
bp	boiling point
br	broad (spectra)
CDCl_3	deuterated chloroform
coe	cyclooctene
d	doublet (spectra)
dd	doublet of doublets (spectra)
ddd	doublet of doublet of doublets (spectra)
DNA	deoxyribonucleic acid
FT	Fourier transform
g	gram(s)
h	hour(s)
Hz	hertz
IR	infrared
J	coupling constant
m	multiplet (spectra)
mg	milligram(s)
MHz	megahertz
mmol	millimole(s)
mp	melting point

NMR	nuclear magnetic resonance
N ₂	dinitrogen (inert environment)
ov	overlapping (spectra)
ppm	parts per million (NMR)
RT	room temperature
s	singlet (spectra)
t	triplet (spectra)

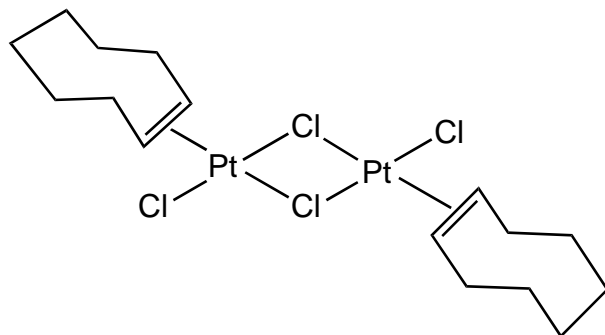
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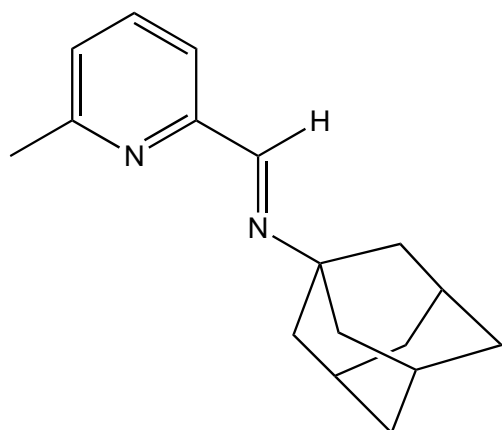
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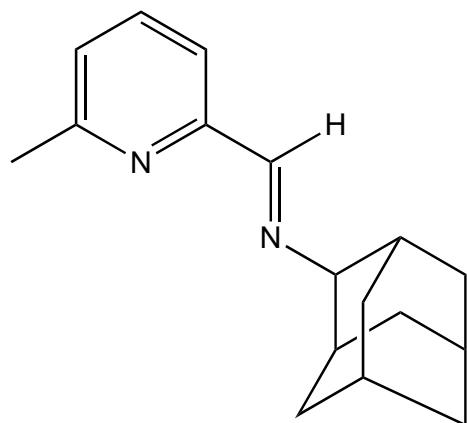
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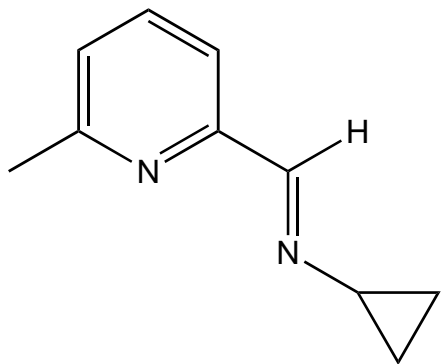
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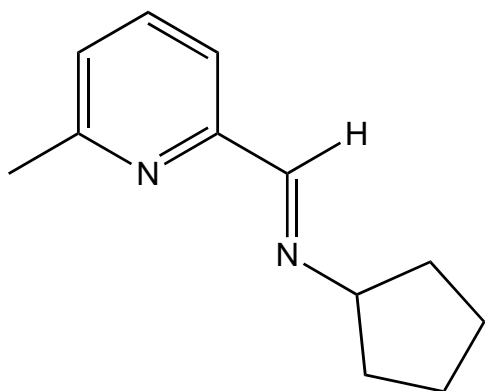
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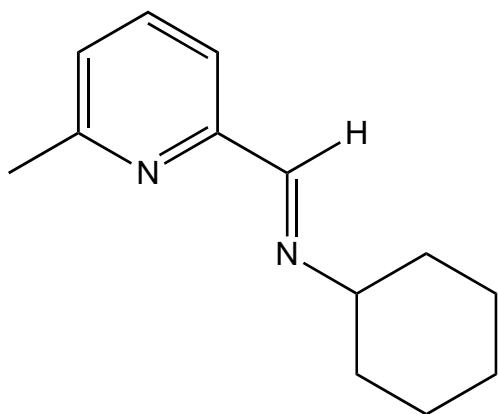
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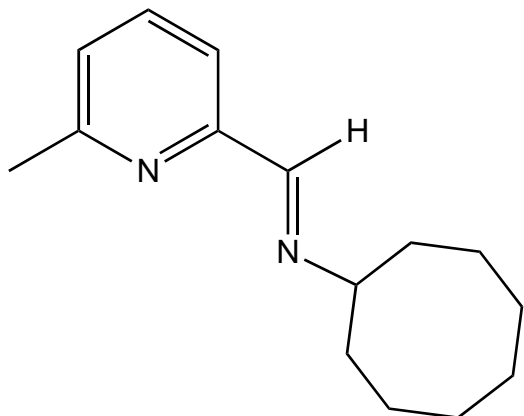
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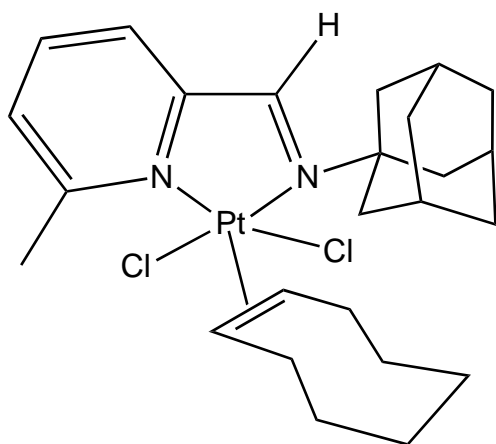
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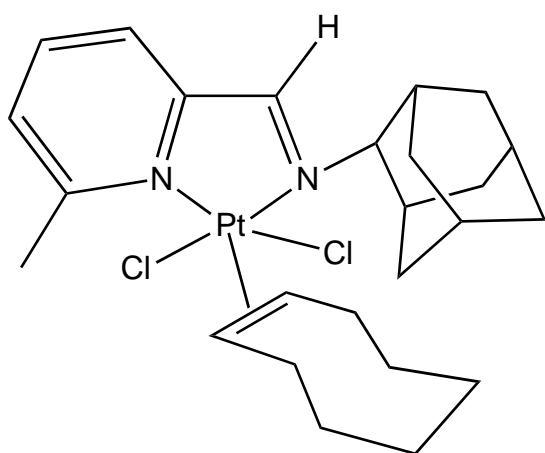
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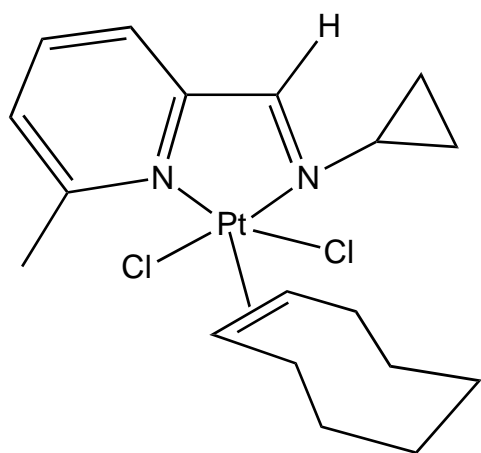
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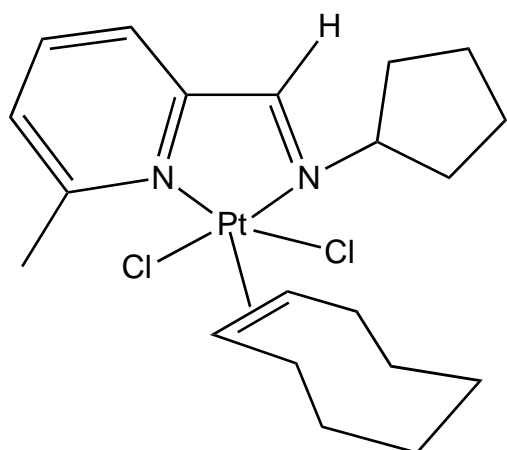
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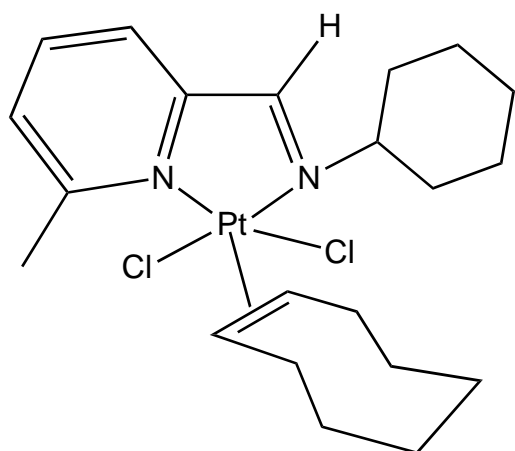
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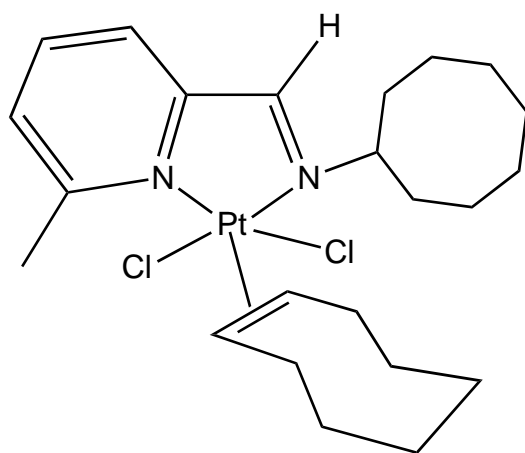
Compound 2c



Compound 2d



Compound 2e



Compound 2f

Introduction

1.1 A Synopsis of Cancer

Cancer continues to be a leading health related cause of death all around the world.¹ Approximately 19.3 million cancer cases were recorded in the year 2020 with an estimated number of 10 million deaths worldwide.¹ Over the years, innumerable research studies have been conducted in attempts to obtain a favorable prognosis.² However, even with the recent advancements in technology and new treatment strategies, cancer continues to be ubiquitous.²⁻³ Moreover, late diagnosis and disease recurrence further contributes to the high mortality rates associated with different types of cancer.⁴

Cancer is characterized by the unrestrained and uncontrolled proliferation of abnormal cells caused by mutated genes.⁵ This results in the spontaneous aggregation of cancer cells into clusters, leading to the growth of a tumor.⁵⁻⁶ Cancer cells also have the tendency to become malignant, allowing them to progressively invade surrounding tissues and form new tumors *via* the process of metastasis.⁶ Cancer is generally associated with inherent and environmental factors, which are the root causes of development of cancer in the human body.⁷

Treatment options such as chemotherapy, radiation therapy, and surgery are available for a wide range of cancers.⁸ Among them, chemotherapy is one of the most

effective treatments.⁹ Platinum-containing coordination complexes such as cisplatin and its derivatives have been widely used as chemotherapeutic drugs.⁹ Chemoresistance occurs when cancer cells stop responding to chemotherapeutic drugs with the same efficacy as before and become resistant to the drug.⁹ For decades, it has become increasingly popular to conduct research on platinum coordination complexes to discover improved anticancer drugs.

1.2 Platinum-based Cancer Therapy

Platinum is a transition metal that forms coordination complexes with oxidation states of +2 and +4.¹⁰ The mechanistic approach of platinum(II)-based drugs relates to their ability to bind to the nitrogenous bases on DNA to form adducts.¹⁰ According to the Hard Soft Acid Base (HSAB) theory, platinum(II) is considered as a soft Lewis acid that prefers to form coordinate bonds with highly polarizable soft Lewis bases.¹⁰⁻¹² It is assumed that platinum(II) being a soft Lewis acid firstly react with sulfur-containing biomolecules, which is kinetically more favorable.^{11,12} Subsequently, platinum(II) complexes then coordinate to DNA to form thermodynamically more favorable platinum-DNA adducts.¹¹

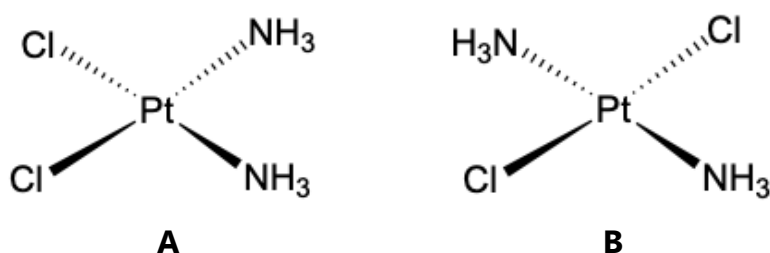


Figure 1. Chemical structure of A) cisplatin and B) transplatin

One of the most widely used platinum(II)-based complexes for cancer treatment is cisplatin $[\text{PtCl}_2(\text{NH}_3)_2]$ (Fig. 1).¹³ Cisplatin exhibits a square planar coordination geometry while maintaining a d^8 electron configuration, which contributes to the stability of the compound.¹⁴ As demonstrated in Figure 1A, the platinum center of cisplatin coordinates to two ligands of chloride in a *cis* configuration. On the other hand, its geometric isomer, transplatin, is proven to be clinically ineffective (Fig. 1B).¹⁵ Transplatin is highly reactive and is rapidly deactivated in blood circulation before its delivery to the target site.¹⁵

1.3 An Overview of Cisplatin

The antineoplastic properties of cisplatin was accidentally discovered in 1965 by Barnett Rosenberg, who was interested in investigating the effects of electric fields on cell division in *Escherichia coli*.¹³ It was later found that cisplatin inhibits cell division and became the first platinum-based anticancer drug to be approved by the Food and Drugs Administration (FDA) for clinical use.¹³ Since then, it has successfully improved the prognosis of several cancers, including testicular, ovarian, neck, and lung cancer.¹³ Cisplatin's discovery was a breakthrough in the drug development for cancer treatment and today, it is widely used as a common chemotherapeutic regimen in combination therapies to enhance chemotherapy treatments.^{13,15} Although cisplatin has demonstrated improved efficacy against cancer cells, it has been linked to various toxic side effects such as nausea and nephrotoxicity due to its tendency to react with sulphur-

containing groups.¹⁵ Moreover, its inclination towards chemoresistance has been a major impediment against platinum-based treatment.¹⁵

1.3.1 Cisplatin Mechanism of Action

Cisplatin is administered intravenously to patients, after which it is transported through the bloodstream.¹³ It remains a neutral molecule as the high chloride concentration (~100 mM) in the extracellular environment prevents cisplatin from being hydrolyzed.¹⁴ Due to no net charge, cisplatin can diffuse through the lipid bilayer matrix of the cell membrane *via* passive diffusion.¹⁶ Subsequently, evidence suggests that uptake of cisplatin is also facilitated by copper transporters, such as CTR1, and organic cation transporters, such as hOCT.¹⁶ Once it enters the cell, cisplatin undergoes the process of aquation due to low intracellular chloride levels (~4 mM), resulting in one (monoqua) or both (diaqua) of the chloride ligands being displaced by water (Fig. 2).¹⁴ The hydrolyzed form of cisplatin act as a potent electrophile to react with nucleophilic sites, including the nitrogenous bases in DNA.¹⁴

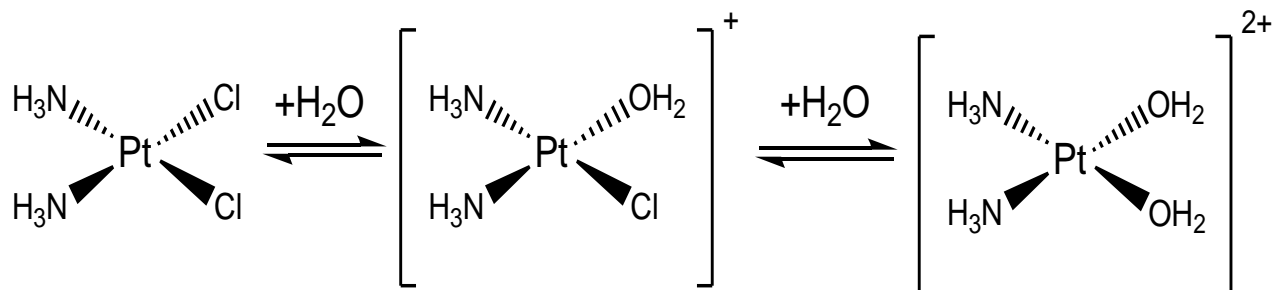


Figure 2. Hydrolysis of cisplatin into monoqua and diaquated forms

The principal mechanism of action of cisplatin is characterized by the formation of adducts upon its interaction with cellular DNA.¹⁶ Cisplatin binds to the nitrogenous bases on DNA, particularly at the N7 position on the purine residues with a higher preference to bind with a guanosine over an adenosine.¹⁶⁻¹⁷ This preference is due to the N1 site on guanine being protonated, resulting in greater localization of electron density on the N7 atom (Fig. 3).¹⁷ On the other hand, adenine has an exposed N1 lone pair, thus delocalizing the electron density to the entire molecule.¹⁷ For this reason, cisplatin will more likely bind to guanine as it is more nucleophilic than adenine and form either monofunctional or bifunctional adducts (Fig. 4).

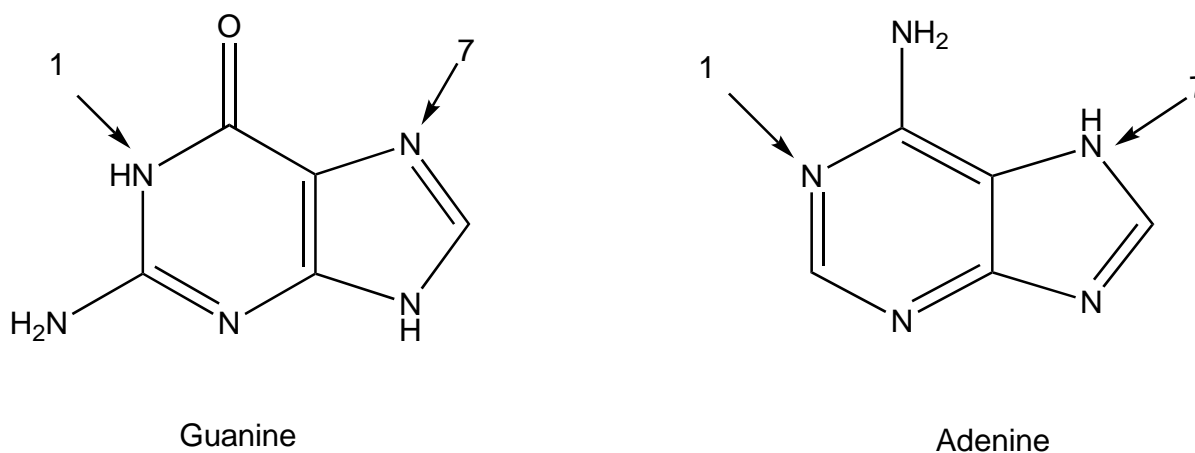


Figure 3. N1 and N7 sites on guanine and adenine bases

Bifunctional adducts, including 1,2-GG and 1,2-GA intrastrand crosslinks, account for ~90% of all adducts formed by cisplatin.¹⁰ Interstrand, monofunctional, and 1,3-intrastrand crosslinks only account for a small percentage of DNA-cisplatin adducts.¹⁰ This interaction of cisplatin and DNA results in the distortion to the double helical

structure, blocking replication and transcription.^{10,17} In efforts to repair the damage, the DNA adducts trigger a series of repair pathways, including nucleotide excision repair (NER) and mismatch repair (MMR), that ultimately induce apoptotic signaling pathways.¹⁰

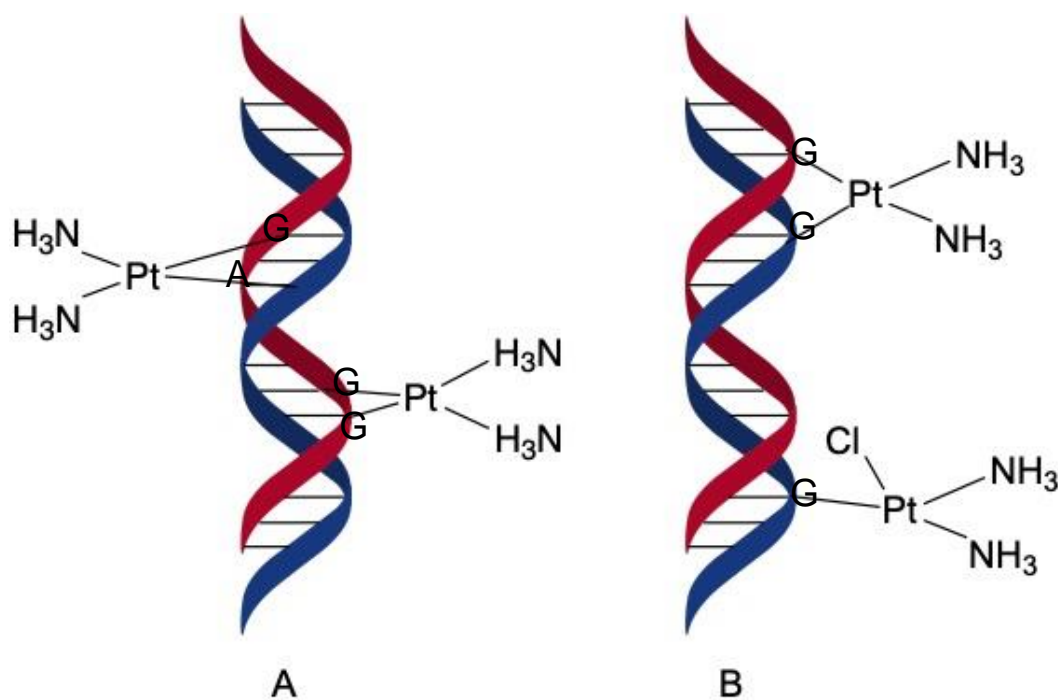


Figure 4. DNA adducts formed by cisplatin. (A) 1,2- and 1,3-intrastrand crosslinks and (B) interstrand and monoadduct crosslinks.

1.3.2 Cisplatin-induced toxicity

Due to lack of selectivity to target tumor tissue, cisplatin cannot discriminate between tumor cells and normal cells.²¹ As such, cisplatin-based therapy is linked to numerous undesirable side effects such as nephrotoxicity, nausea, and vomiting.²¹ The risk of patients acquiring nephrotoxicity or renal toxicity is between 20% to 35%.²²

Cisplatin can also induce acute kidney injury (AKI) in cancer patients that can lead to death.²² Some common outcomes of kidney dysfunction include decreased renal plasma flow, decline in glomerular filtration rate, and elevated serum creatinine.²²

Neurotoxicity is another side effect prevalent in patients undergoing cisplatin-based therapy.²³ Cisplatin-induced neurotoxicity includes peripheral neuropathy and ototoxicity (hearing loss and tinnitus). Approximately 50% of the patients develop signs of neurotoxicity including loss of position sense, weakness, tremor, and loss of taste.²³ Ototoxicity is the result of damage to the hair cells of the cochlea caused by oxidative stress and reduced L-glutathione concentrations.²³

Cisplatin-induced myelosuppression is also a major side effect that is caused by decreased bone-marrow function resulting in depleted blood cell count.²⁴ Common signs and symptoms include fever and increased risk of developing infections.²⁴ Many other adverse side effects are attributed to cisplatin-based therapy. As such, search for improved platinum-based anticancer drugs exhibiting reduced toxicity is ongoing. However, drug resistance is another major concern with cisplatin treatment.

1.3.3 Cisplatin Resistance

Chemoresistance is a major impediment against the clinical use of cisplatin.¹⁸ Tumors exhibit resistance to cisplatin either intrinsically or as acquired during the course of cisplatin-based therapy.¹⁸ A plethora of tumor resistance mechanisms associated with cisplatin-based chemotherapy have emerged (Fig. 5). Major mechanisms include

decreased drug import, increased drug export, increased inactivation of cisplatin by thiol-containing molecules, increased DNA damage repair, and failure of initiating apoptotic signaling pathways.^{21,25} More than one mechanism may be simultaneously involved in the resistance.

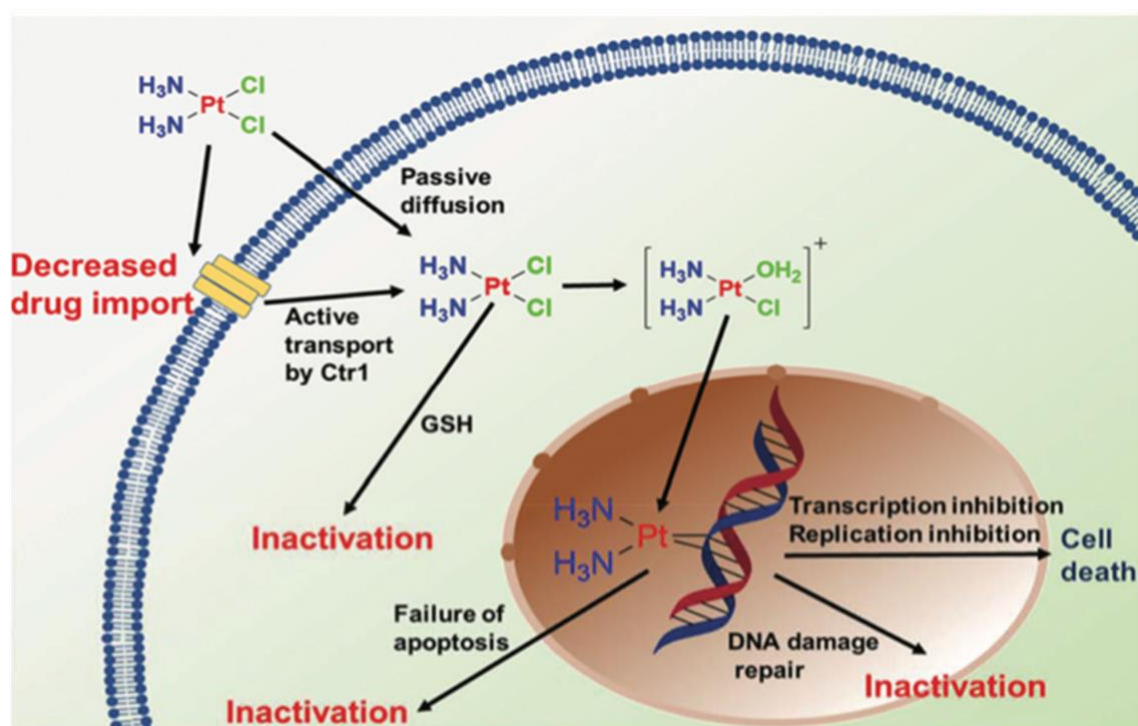


Figure 5. Schematic representation of resistance pathways of cisplatin. Decreased drug import, increased drug export, increased inactivation of cisplatin, increased DNA damage repair, and failure of apoptosis are major mechanisms resulting in cisplatin resistance.²¹

Decrease in cellular uptake of cisplatin is due to the lower expression of the copper transporter CTR1, a major influx transporter responsible for the uptake of cisplatin, resulting in cisplatin resistance.^{18,25} Studies have also indicated the presence of efflux proteins (ATP7A and ATP7B) that regulate the cellular outflow of cisplatin.²⁶ High

expressions of these two ATPases correspond to increase in efflux as typically seen during resistance.²⁶ Additionally, the activated aqua cisplatin species is more prone to bind to cytoplasmic thiol-containing molecules such as glutathione (GSH) and metallothionein (MT).^{25,26} Increased GSH and MT levels have also been associated with resistance as it results in the inactivation of cisplatin.²⁵

DNA repair by the nucleotide-excision repair (NER) pathway is another factor contributing towards cisplatin resistance.²⁵⁻²⁷ The NER endonuclease protein ERCC1 (excision repair cross-complementing-1) plays a critical role for the excision of platinum adducts on the DNA strand.^{25,26} In ovarian cancer cell lines, increased expression levels of ERCC1 is associated with enhanced repair of cisplatin-induced DNA damage, thus contributing to resistance.²⁶ Cisplatin resistance is also linked to translesion synthesis, a DNA damage tolerance mechanism, whereby specialized DNA polymerases (polymerase β and η) can bypass cisplatin-DNA adducts.^{18,27} As such, the polymerases can continue replicating DNA regardless of the cisplatin-induced DNA damage.²⁷

Drug resistance and toxicity are two major concerns associated with cisplatin-based therapy.¹⁸ To make platinum-based chemotherapy safer for cancer patients, several efforts have been made to design improved anticancer drugs. Combination therapies of cisplatin with other anticancer drugs is also an approach used to overcome drug resistance and reduce toxicity.¹⁸

1.3.3 Analogues of Cisplatin

In attempts to overcome the drawbacks of cisplatin, much effort has been put into developing new platinum-based drugs. Several second- and third-generation platinum compounds are also widely accepted as anticancer drugs, including carboplatin and oxaliplatin.^{10,18} In 1989, carboplatin (*cis*-diammine-[1,1-cyclobutanedicarboxylato] platinum(II)) was approved by the FDA for ovarian cancer treatment.¹⁸ It is used as an alternative drug in chemotherapy treatments as it is associated with a lower toxicity profile than its predecessor, cisplatin.^{10,15} The greatest advantage of carboplatin is that it exhibits reduced nephrotoxic effects as compared to cisplatin, due to its slower rate of aquation.¹⁵ Its chemical structure is relatively distinct from cisplatin, carrying a bidentate carboxylate ligand instead of the chloride atoms which offers greater stability (Fig. 6a).¹⁵ However, due to the retention of the same non-leaving group ligands, carboplatin shows cross-resistance with cisplatin.¹⁴ Moreover, one of the major drawbacks associated with carboplatin-based chemotherapy is that it is significantly less potent or less effective than cisplatin.¹⁵ Due to this reason, carboplatin must be administered at much higher doses than cisplatin.¹⁰ Dose-limiting myelosuppression is another major concern following carboplatin-based chemotherapy.¹⁸

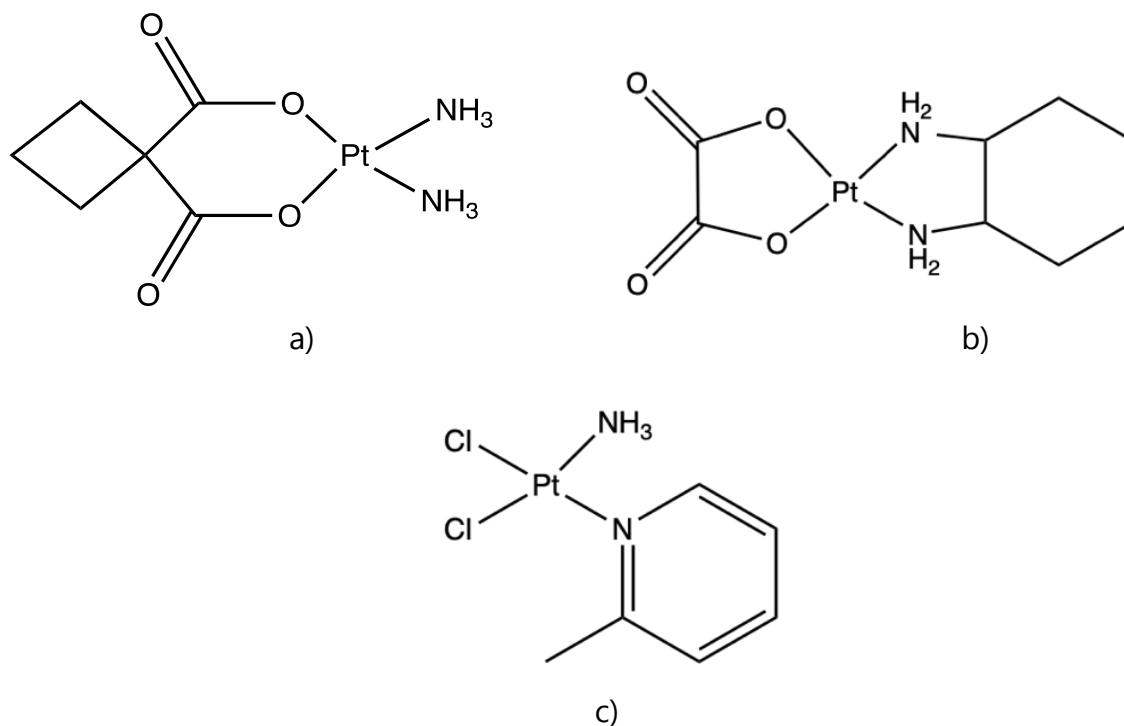


Figure 6. Chemical structures of (a) carboplatin, b) oxaliplatin, c) picoplatin

Another analogue of cisplatin that has emerged as a prominent anticancer agent, mostly for the treatment of colorectal cancer, is oxaliplatin (Fig. 6b).¹⁸ Oxaliplatin is often used in cancer treatments as it offers better tolerability in addition to a lower toxicity profile than both cisplatin and carboplatin.^{10,18} In 2004, it was approved by the FDA for the treatment of adjuvant and metastatic colorectal cancer.¹⁸ Oxaliplatin is administered intravenously and is commonly used in combination with fluorouracil and leucovorin in patients with colorectal cancer.¹⁸ It is characterized by the presence of oxalate and diaminocyclohexane ligands (DACH) instead of the amine groups as seen in cisplatin.¹⁰ Studies show that the DACH ligands contribute to the increased lipophilicity of

oxaliplatin, resulting in its improved passive uptake.¹⁷ The dose-limiting toxicities of oxaliplatin includes neurotoxicity, nausea, and vomiting.¹⁸

Picoplatin (*cis*-amminedichloro[2-methylpyridine] platinum(II)), also known as ZD0473, is another cisplatin analogue that had broadened the clinical use of platinum-based chemotherapy (Fig. 6c).^{28,29} Picoplatin was designed to provide increased steric hinderance around the platinum center in attempts to reduce inactivation by thiol containing species such as GSH and MT.³⁰ The 2-methylpyridine ring lies perpendicular to the plane of the platinum ligands, positioning the methyl group above the platinum center.³¹ This orientation of the methyl group adds steric bulk to the platinum atom, decreasing the rates of hydrolysis and substitution reactions of picoplatin.^{30,31} As such, this allows enhanced binding of picoplatin to DNA and thus demonstrates decreased toxic side effects.^{28,29} Clinical trials involving picoplatin revealed that it displayed promising clinical activity in patients with small-cell lung cancer.^{18,29} Compared with cisplatin, picoplatin is reported to exhibit reduced nephrotoxic and neurotoxic side effects with myelosuppression being a dose-limiting factor.¹⁸

1.4 A New Design Strategy

Several picoplatin analogues using iminopyridine ligands containing primary bulky amines have been previously reported by the Westcott group. The group particularly focused on synthesizing platinum complexes that did not contain the methyl substituent as seen in picoplatin.³⁰ Alternatively, the current study focuses on synthesizing a series of novel platinum(II) complexes containing bidentate iminopyridine ligands that contain a 6-methyl substituent. Figure 6 shows the general structure of platinum(II) complexes reported in this study.

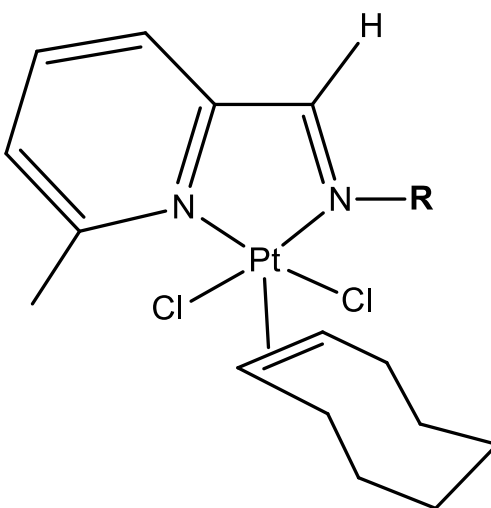


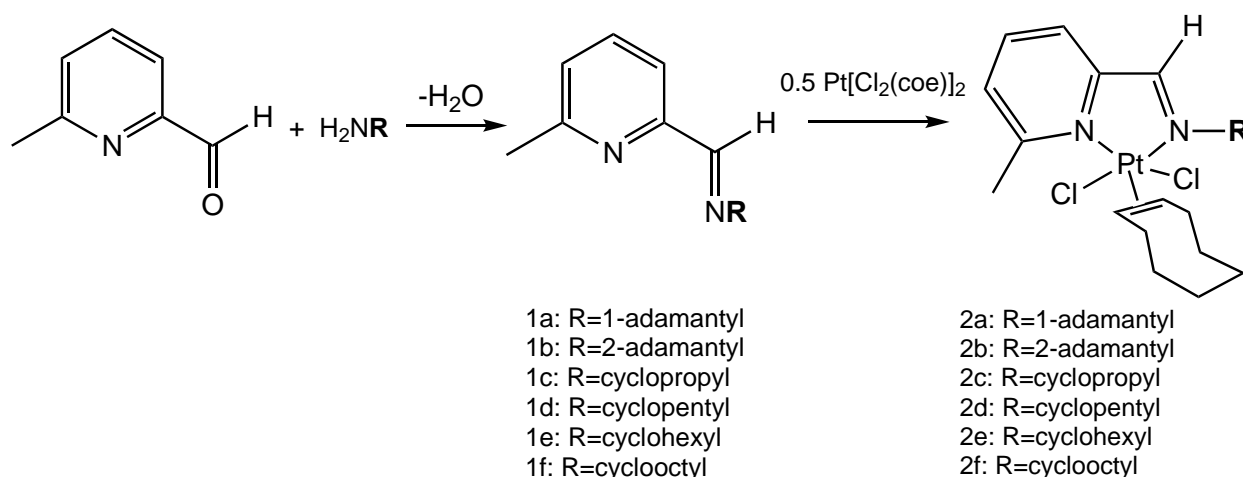
Figure 6. General structure of platinum(II) complexes synthesized with the addition of iminopyridine ligands to $[\text{PtCl}_2(\text{coe})]_2$.

When designing the skeleton of these ligands, incorporating both a bulky side chain into the imino moiety (Figure 6, R) and a 6-methyl substituent on the iminopyridine ring is of particular interest as it may provide steric congestion around the

platinum center.³⁰ Steric hinderance is believed to shield the metal center while slowing down the rate of hydrolysis and ligand substitution reactions of the Pt(II) complexes.³⁰ This may potentially allow higher selectivity in binding to DNA.³⁰ Accordingly, these complexes are designed to reduce some of the adverse side effects associated with cisplatin.

Objective

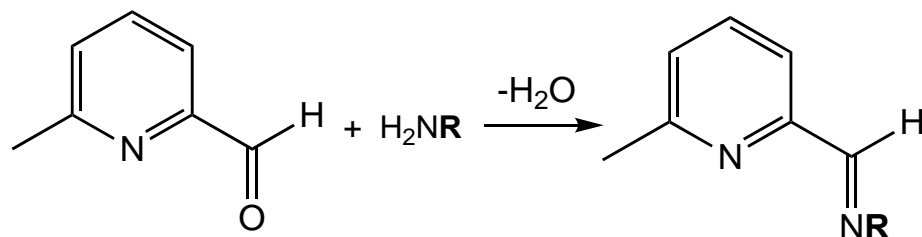
The goal of this study is to synthesize and characterize six novel platinum(II) complexes by the addition of methylated bidentate iminopyridine ligands derived from 6-methyl-2-pyridinecarboxylaldehyde and bulky cycloalkyl amines (Scheme 2). All platinum(II) coordination complexes will be characterized using FT-IR spectroscopy, multinuclear NMR spectroscopy, and melting point analysis. The bulky cycloalkyl groups and the methyl group substituent are expected to induce steric hinderance around the platinum center. It is anticipated that the incorporation of the steric bulk will prevent degradation of Pt(II) complexes from biomolecules, slow drug aquation, and presumably achieve enhanced selectivity in binding to DNA.



Scheme 1. General synthetic pathway for the synthesis of iminopyridine ligands and platinum(II) complexes.

Results and Discussion

3.1 Iminopyridine Ligand Synthesis



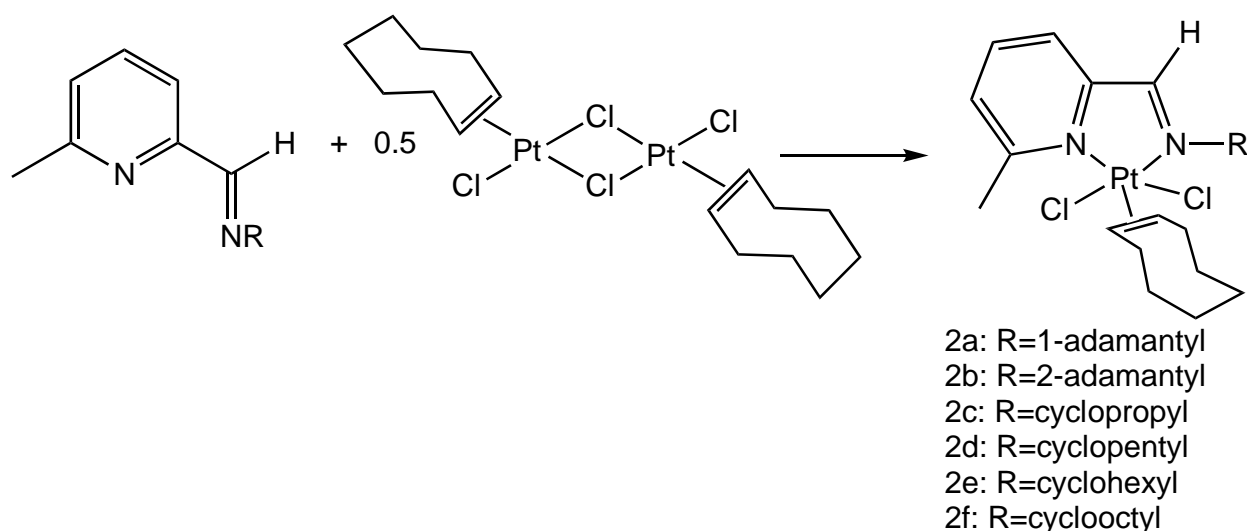
- 1a: R=1-adamantyl
- 1b: R=2-adamantyl
- 1c: R=cyclopropyl
- 1d: R=cyclopentyl
- 1e: R=cyclohexyl
- 1f: R=cyclooctyl

Scheme 2. Synthesis of iminopyridine ligands *via* condensation reaction

Six methylated iminopyridine ligands were synthesized *via* a simple condensation reaction of 6-methyl-2-pyridinecarboxaldehyde and the corresponding primary amines in dichloromethane (Scheme 2). The reaction proceeds with a nucleophilic attack at the carbonyl carbon of the aldehyde by the amino nitrogen of the amine. Due to the compounds being air-sensitive, all experiments were performed in the glovebox under dinitrogen atmosphere. Molecular sieves were added to the reaction mixture as water is produced as a by-product during imine formation. The reaction was left to stand for five days at room temperature before the solvent was removed under vacuum to afford the desired products in moderate to high yields (49%-85%).

Compounds **1a-f** were characterized by a number of physical methods, including ^1H NMR and ^{13}C $\{^1\text{H}\}$ NMR spectroscopy and FT-IR spectroscopy. The ^1H resonances of the imine ligands were observed, with the aromatic H-atoms appearing at 7.14-7.84 ppm and the imine protons appearing as a singlet at 8.31-8.39 ppm. The absence of the aldehyde peak at 10 ppm and the presence of the imine peak indicated the formation of the imine. The IR spectra was obtained for further confirmation of reaction completion. The appearance of a medium peak at 1645 cm^{-1} indicated the presence of the carbon nitrogen double bond ($\nu(\text{C}=\text{N})$). Two of the iminopyridine ligands obtained, **1a** and **1b**, were solids with melting point ranges of $52.9\text{-}54.2\text{ }^\circ\text{C}$ and $102.3\text{-}103.0\text{ }^\circ\text{C}$, respectively.

3.2 Platinum Complex Synthesis



Scheme 3. Synthesis of platinum(II) complexes **2e-f**

After successful synthesis, appropriate iminopyridine ligands were coordinated to the stable organometallic dimer $[\text{PtCl}_2(\text{coe})]_2$ (coe = *cis*-cyclooctene) as illustrated in

Scheme 3. The appropriate imine ligand was added dropwise to a half equivalent of $[\text{PtCl}_2(\text{coe})]_2$. The reaction mixture was left to stir for 18h at RT in the glovebox. The desired platinum complexes were afforded in low to high yields (23%-96%) following the removal of solvent by rotary evaporation.

All platinum(II) compounds were analyzed using FT-IR, multinuclear NMR spectroscopy, and melting point determination. Upon complexation of the ligand to the metal center, platinum satellite peaks at 8.80-8.96 ppm were observed with coupling constants ranging from 15Hz to 18Hz in the ^1H NMR spectra. The broad peak at 3.94-4.15 ppm with platinum satellite peaks ($J_{\text{HPt}} = 40 \text{ Hz}$) indicated the presence of a bound cyclooctene group to the metal center. Moreover, an extra peak at $\sim 71 \text{ ppm}$ indicating the presence of cyclooctene group was reported in the ^{13}C NMR spectra. The compounds were dissolved in hexane in attempts to remove the bound coe, however it remained attached. It was assumed that the resulting compounds were five-coordinated Pt(II) complexes that exhibited a trigonal bipyramidal geometry with two chloride groups, two nitrogen atoms, and a cyclooctene group attached to the platinum center. Though five-coordinated Pt(II) complexes represent a minority of platinum(II) compounds, previous studies such as Renzi, *et. al.* and Annuziata, *et. al.* have reported similar compounds.^{32,32} X-ray crystallography should be performed in the future to confirm their solid-state structures.

Finally, melting point determination was performed on all platinum complexes, which ranged from 135-188 °C. The IR spectra revealed that the iminopyridine ligand is coordinated to the platinum, which is indicated by the decrease in frequency of the carbon nitrogen double bond peak. Due to time constraints, elemental analysis was performed on only three of the complexes- **2a**, **2e**, and **2f**.

Conclusions and Future Directions

For this project, six platinum(II) complexes based on the structural motif of picoplatin were successfully synthesized. Iminopyridine ligands containing bulky cycloalkyl amines were synthesized *via* a condensation reaction. The resulting iminopyridine ligands were subsequently coordinated to platinum following the addition of $[\text{PtCl}_2(\text{coe})]_2$. All complexes were fully characterized using FT-IR, NMR spectroscopy, and melting point determination. Elemental analysis was performed on platinum complexes **2a**, **2e**, and **2f**. X-ray diffraction study must be conducted to support the existing characterization data. Future studies should complete elemental analysis studies and carry out biological testing to test for antineoplastic activity.

Experimental

5.1 General Procedure

Reagents and solvents were obtained from Aldrich chemicals. $[\text{PtCl}_2(\eta^2\text{-coe})]_2$ (coe = *cis*-cyclooctene) was prepared according to the procedure outlined in Shaver *et al.*³⁴ ^1H NMR and $^{13}\text{C}\{^1\text{H}\}$ NMR spectra were recorded in CDCl_3 with a Varian Mercury 200 Plus spectrometer. Multiplicities are reported as (s) singlet, (d) doublet, (t) triplet, (m) multiplet, (br) broad, and (ov) overlapping. The chemical shifts (δ) are reported in ppm and the coupling constant (J) in hertz. FT-IR spectra were recorded on a Nicolet iS5 FT-IR spectrometer and reported in cm^{-1} . Melting points were measured uncorrected with a Stuart SMP30 Mel-Temp apparatus. Reactions were performed under an atmosphere of nitrogen in a MBraun LabMaster glovebox.

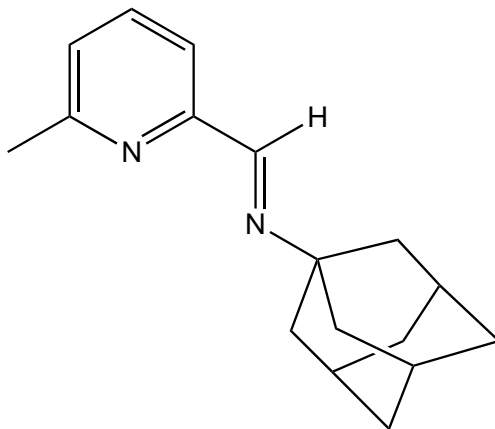
5.2 Amine Synthesis

5.2.1 *Synthesis of 2-adamantylamine for 1b*

2-Adamantylamine hydrochloride (200 mg) was added to a solution of sodium hydroxide (43 mg) and distilled water (10 mL). The reaction mixture was poured into a separatory funnel with dichloromethane (5 mL). After vigorous mixing, the bottom organic layer was extracted, and the solvent was removed using a rotary evaporator to yield a white solid. Yield: 121 mg (75%); m.p.: 240 °C. NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 2.97 (s, 2H, NH₂), 2.00-1.71 (m, 24H), 1.52 (d, J_{HH} = 11.4 Hz, 10H). FT-IR: 2899 (s), 2848 (s), 1548 (m), 1464 (m), 1449 (m), 1379 (m), 1365 (m), 1352 (m), 1104 (m), 978 (m), 895 (m), 843 (m), 800 (m), 781 (m), 746 (m), 673 (m), 626 (m).

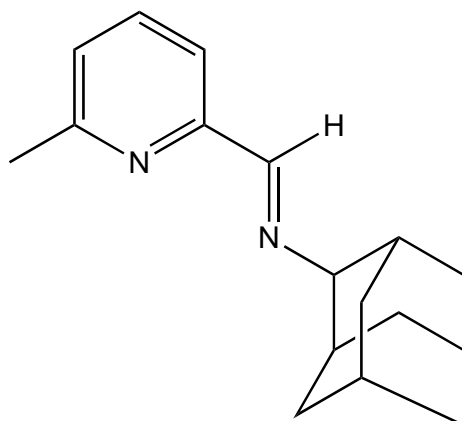
5.3 Synthesis of Iminopyridine Ligands

5.3.1 Synthesis of (*E*)-*N*-((6-methylpyridin-2-yl)methylene)adamantan-1-amine - **1a**



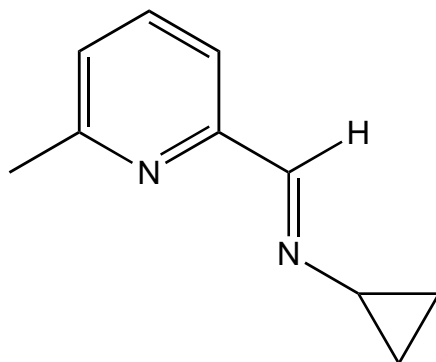
1-Adamantylamine (625 mg, 4 mmol) dissolved in CH₂Cl₂ (5 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (500 mg, 4 mmol) and CH₂Cl₂ (4 mL). Activated molecular sieves (4Å, 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a white solid. Yield: 889 mg (85%). m.p.: 52.9-54.2 °C. NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.33 (s, 1H, HC=N), 7.88 (d, J_{HH} = 8 Hz, 1H, Ar), 7.64 (t, J_{HH} = 8 Hz, 1H, Ar), 7.16 (d, J_{HH} = 8 Hz, 1H, Ar), 2.58 (s, 3H, CH₃), 2.16 (s, 4H), 1.71 (br dd, J_{HH} = 2, 2.2 Hz, 13H). ¹³C{¹H} δ: 157.7, 156.6, 155.2, 136.7, 123.9, 123.9, 117.7. FT-IR: 2910 (m), 2898 (m), 2847 (m), 1642 (m, ν_{CN}), 1588 (m), 1570 (m), 1463 (m), 1452 (m), 1369 (m), 1352 (m), 1340 (m), 1307 (m), 1296 (s), 1253 (s), 1220 (s), 1116 (s), 1101 (s), 1090 (s), 1080 (s), 1043 (s), 991 (s), 983 (s), 929 (s), 814 (m), 805 (m), 786 (m), 733 (s), 717 (s), 657 (s), 643 (s).

5.3.2 Synthesis of (*E*)-*N*-((6-methylpyridin-2-yl)methylene)adamantan-2-amine -**1b**



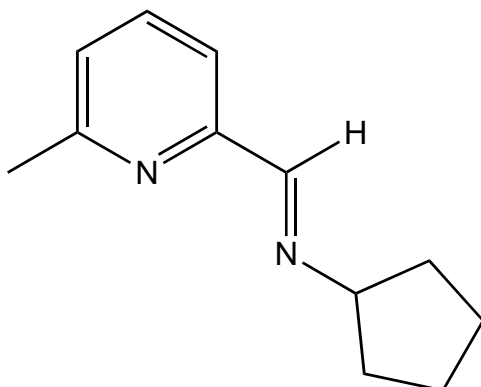
2-Adamantylamine (150 mg, 4 mmol) dissolved in CH₂Cl₂ (5 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (96 mg, 4 mmol) and CH₂Cl₂ (4 mL). Activated molecular sieves (4Å, 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a white solid. Yield: 68 mg (81%). m.p.: 102.3-103.0 °C NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.40 (s, 1H, HC=N), 7.94 (d, J_{HH} = 8 Hz, 1H, Ar), 7.61 (t, J_{HH} = 8 Hz, 1H, Ar), 7.15 (d, J_{HH} = 8 Hz, 1H, Ar), 3.52 (s, 1H), 2.58 (s, 3H, CH₃), 2.37 (d, J_{HH} = 12 Hz, 2H), 1.83-1.56 (br ov m, 12H). ¹³C{¹H} δ: 159.3, 157.7, 154.9, 136.6, 123.9, 117.9, 77.2, 74.1, 38.0, 37.2, 35.3, 32.0, 28.2, 27.3, 24.3. FT-IR: 2899 (s), 2849 (m), 2821, 1646 (m, ν_{CN}), 1590 (m), 1574 (m), 1465 (m), 1446 (m), 1368, 1111 (m), 988 (m), 958 (m), 928 (m), 819 (m), 793 (s), 736 (m), 684 (m), 658 (m), 623 (m).

5.3.3 Synthesis of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclopropanamine -1c



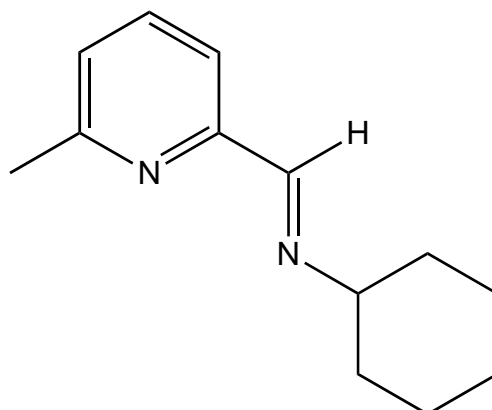
Cyclopropanamine (234 mg, 4 mmol) dissolved in CH_2Cl_2 (5 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (500 mg, 4 mmol) and CH_2Cl_2 (5 mL). Activated molecular sieves (4\AA , 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a dark orange oil. Yield: 361 mg (55%). NMR spectroscopic data (CDCl_3 , ppm): ^1H δ : 8.32 (s, 1H, $\text{HC}=\text{N}$), 7.82 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.60 (t, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.16 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 3.46 (m, 1H), 2.66 (s, 3H, CH_3), 1.88-1.57 (br ov m, 14H, $-(\text{CH}_2)_7-$). $^{13}\text{C}\{^1\text{H}\}$ δ : 159.0, 157.8, 154.5, 136.6, 123.9, 118.1, 71.2, 33.8, 27.2, 25.7, 24.3, 24.0. FT-IR: 2918 (m), 2849 (m), 1713 (s), 1645 (m, ν_{CN}), 1590 (m), 1573 (m), 1455 (m), 1374 (m), 1264 (s), 1221 (m), 1151 (s), 1122 (s), 1084 (s), 1062 (s), 985 (s), 958 (s), 921 (s), 789 (m), 735 (s), 702 (s), 644 (s).

5.3.4 Synthesis of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclopentanamine -1d



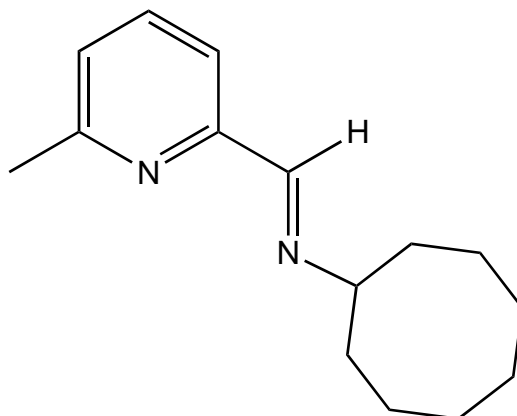
Cyclopentylamine (352 mg, 4 mmol) dissolved in CH₂Cl₂ (4 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (500 mg, 4 mmol) and CH₂Cl₂ (4 mL). Activated molecular sieves (4Å, 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a brown oil. Yield: 575 mg (74%). NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.35 (s, 1H, HC=N), 7.84 (d, J_{HH} = 8 Hz, 1H, Ar), 7.64 (t, J_{HH} = 8 Hz, 1H, Ar), 7.17 (d, J_{HH} = 8 Hz, 1H, Ar), 3.86 (m, 1H), 2.58 (s, 3H, CH₃), 1.94-1.63 (br ov m, 9H, -(CH₂)₄-). ¹³C{¹H} δ: 159.9, 157.9, 154.4, 136.6, 124.0, 118.2, 71.5, 34.3, 24.7. FT-IR: 2955 (m), 2865 (m), 1644 (m, ν_{CN}), 1589 (m), 1572 (m), 1454 (m), 1367 (m), 1315 (m), 1250 (s), 1150 (s), 1084 (s), 985 (s), 967 (s), 919 (s), 790 (m), 736 (s), 658 (s), 632 (s).

5.3.5 Synthesis of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclohexanamine -1e



Cyclohexylamine (398 mg, 4 mmol) dissolved in CH_2Cl_2 (4 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (500 mg, 4 mmol) and CH_2Cl_2 (5 mL). Activated molecular sieves (4A°, 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a pale-yellow oil. Yield: 394 mg (49%). NMR spectroscopic data (CDCl_3 , ppm): ^1H δ : 8.37 (s, 1H, $\text{HC}=\text{N}$), 7.83 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.65 (t, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.17 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 3.27 (m, 1H), 2.66 (s, 3H, CH_3), 1.87-1.20 (br ov m, 11H, $-(\text{CH}_2)_5$). $^{13}\text{C}\{^1\text{H}\}$ δ : 159.9, 157.9, 154.4, 136.7, 124.1, 118.2, 69.6, 34.1, 25.6, 24.7, 24.4. FT-IR: 2926 (m), 2852 (m), 1645 (m, ν_{CN}), 1590 (m), 1571 (m), 1450 (m), 1372 (m), 1348 (m), 1331 (s), 1298 (s), 1251 (s), 1222 (s), 1147 (s), 1076 (s), 988 (s), 964 (s), 887 (s), 845 (m), 790 (m), 736 (s), 652 (s).

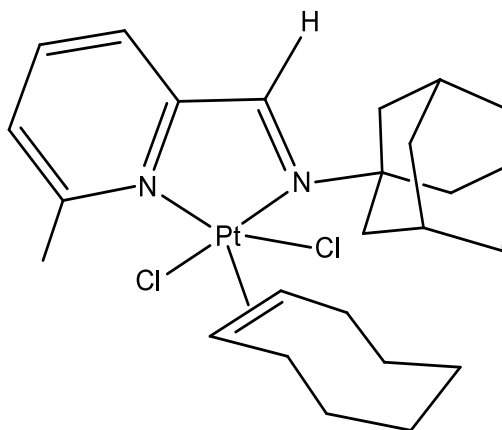
5.3.6 Synthesis of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclooctanamine -1f



Cyclooctanamine (517 mg, 4 mmol) dissolved in CH_2Cl_2 (4 mL) was added dropwise to a stirred solution of 6-methyl-2-pyridinecarboxylaldehyde (515 mg, 4 mmol) and CH_2Cl_2 (5 mL). Activated molecular sieves (4A°, 5 g) were added to the reaction mixture and left to stand for 5 days at RT under nitrogen. The solvent was removed under vacuum to afford a dark orange oil. Yield: 689 mg (70%). NMR spectroscopic data (in CDCl_3 , ppm): ^1H δ : 8.32 (s, 1H, $\text{HC}=\text{N}$), 7.82 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.60 (t, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 7.16 (d, $J_{\text{HH}} = 8$ Hz, 1H, Ar), 3.46 (m, 1H), 2.66 (s, 3H, CH_3), 1.88-1.57 (br ov m, 14H, $-(\text{CH}_2)_7-$). $^{13}\text{C}\{^1\text{H}\}$ δ : 159.0, 157.8, 154.5, 136.6, 123.9, 118.1, 71.2, 33.8, 27.2, 25.7, 24.3, 24.0. FT-IR: 2918 (m), 2849 (m), 1713 (s), 1645 (m, ν_{CN}), 1590 (m), 1573 (m), 1455 (m), 1374 (m), 1264 (s), 1221(m), 1151 (s), 1122 (s), 1084 (s), 1062 (s), 985 (s), 958 (s), 921 (s), 789 (m), 735 (s), 702 (s), 644 (s).

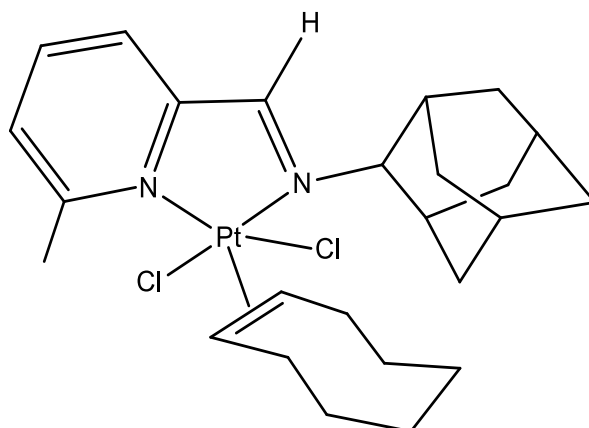
5.4 Synthesis of Platinum Iminopyridine Complexes

5.4.1 Synthesis of compound **2a**



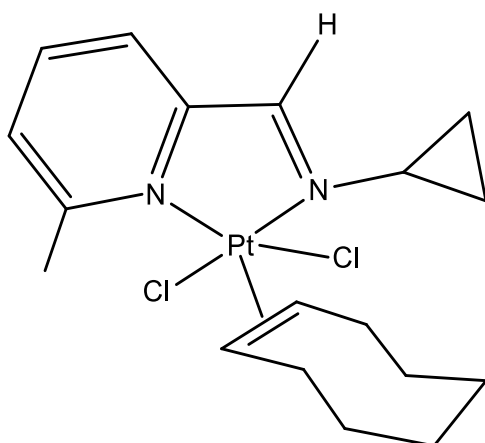
A CH₂Cl₂ (2 mL) solution of **1a** (68 mg, 0.27 mmol) was added to [PtCl₂(coe)]₂ (100 mg, 0.13 mmol) in CH₂Cl₂ (3 mL). The reaction mixture was stirred for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield a bright yellow solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 92 mg (55%); m.p.:186-188 °C. NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.81 (s, *J*_{HPt}= 17 Hz, 1H, HC=N), 7.84 (t, *J*_{HH}= 8 Hz, 1H, Ar), 7.53 (t, *J*_{HH}= 8 Hz, 1H, Ar), 4.15 (br d, *J*_{HPt}= 38 Hz, *J*_{HH}= 11 Hz, 2H), 3.27 (s, 3H, CH₃), 2.30-1.48 (br ov m, 27H). ¹³C{¹H} δ: 157.8, 156.6, 155.2, 136.7, 123.9, 117.7, 58.1, 43.0, 36.5, 29.5, 24.3. FT-IR: 2911, 2848, 1594, 1462, 1448, 1414, 1305, 1161, 1096, 1082, 1013, 951, 922, 742. *Anal.* calc. for C₂₅H₃₆N₂Cl₂Pt • CH₂Cl₂ (630.57 g/mol) (%): C 43.54, H 5.50, N 4.08; found: C 43.65, H 5.35, N 3.91.

5.4.2 Synthesis of compound **2b**



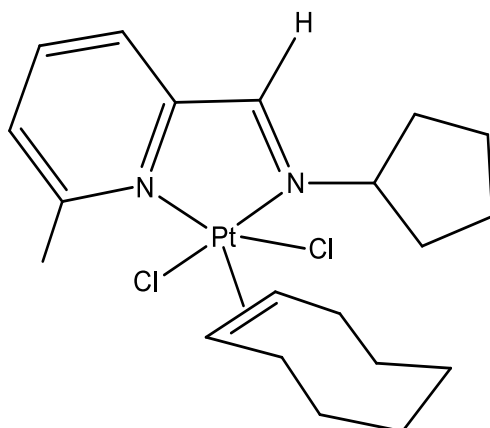
A CH₂Cl₂ (2 mL) solution of **1b** (134 mg, 0.53 mmol) was added to [PtCl₂(coe)]₂ (200 mg, 0.27 mmol) in CH₂Cl₂ (4 mL). The reaction mixture was stirred for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield a yellow solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 161 mg (96%); m.p.: 182-188 °C. NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.81 (s, *J*_{HPt} = 17 Hz, 1H, HC=N), 7.84 (t, *J*_{HH} = 8 Hz, 1H, Ar), 7.53 (t, *J*_{HH} = 8 Hz, 1H, Ar), 4.15 (br d, *J*_{HPt} = 38 Hz, *J*_{HH} = 11 Hz, 2H), 3.27 (s, 3H, CH₃), 2.30-1.48 (br ov m, 27H). ¹³C{¹H} δ: 157.8, 156.6, 155.2, 136.7, 123.9, 117.7, 58.1, 43.0, 36.5, 29.5, 24.3. FT-IR: 2904 (m), 2850 (m), 1620 (m, ν_{CN}), 1596 (m), 1465 (m), 1450 (m), 1380 (m), 1358 (m), 1340 (m), 1318 (m), 1300 (s), 1278 (s), 1246 (s), 1232 (s), 1217 (s), 1098 (m), 1078 (m), 1069 (m), 1055 (m), 1035 (m), 961 (s), 943 (s), 891 (s), 819 (m), 797 (m), 787 (m), 750 (m), 733 (s), 715 (s), 701 (s), 630 (s).

5.4.3 Synthesis of compound **2c**



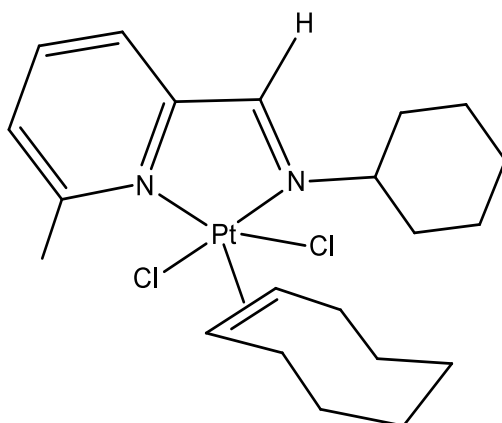
A CH₂Cl₂ (3 mL) solution of **1c** (42 mg, 0.31 mmol) was added to [PtCl₂(coe)]₂ (100 mg, 0.13 mmol) in CH₂Cl₂ (5 mL). The reaction mixture was left to stand for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield a brown solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 132 mg (92%); m.p.: 142-144 °C (decomp.). NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.96 (s, *J*_{HPt} = 15 Hz, 1H, *H*C=N), 7.83 (t, *J*_{HH} = 8 Hz, 1H, Ar), 7.51 (ov d, *J*_{HH} = 8 Hz, 1H, Ar), 4.11 (br d, *J*_{HPt} = 40 Hz, *J*_{HH} = 10 Hz, 2H), 3.23 (s, 3H, CH₃), 2.17-1.27 (br ov m, 25H). ¹³C{¹H} δ: 160.5, 159.6, 150.9, 138.7, 128.0, 124.2, 57.2, 42.4, 28.4, 26.3. FT-IR: 2918 (m), 2852 (m), 1645 (m, *ν*_{CN}), 1597 (s), 1571 (s), 1463 (m), 1445 (m), 1378 (m), 1358 (m), 1278 (s), 1255 (s), 1217 (s), 1161 (s), 1136 (s), 1095 (s), 1063 (s), 1033 (s), 966 (s), 863 (w), 811 (m), 791 (m), 740 (s), 702 (s), 663 (s).

5.4.4 Synthesis of compound **2d**



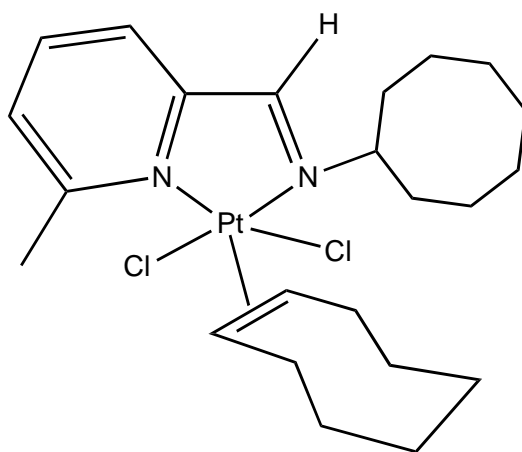
A CH₂Cl₂ (3 mL) solution of **1d** (50 mg, 0.27 mmol) was added to [PtCl₂(coe)]₂ (100 mg, 0.13 mmol) in CH₂Cl₂ (5 mL). The reaction mixture was stirred for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield an orange solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 77 mg (63%); m.p.: 174.4-177.2 °C. NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.84 (s, *J*_{HPt} = 18 Hz, 1H, HC=N), 7.84 (t, *J*_{H-H} = 8 Hz, 1H, Ar), 7.54 (t, *J*_{HH} = 8 Hz, 1H, Ar), 4.14 (br d, *J*_{HPt} = 47 Hz, *J*_{HH} = 11 Hz, 2H), 3.25 (s, 3H, CH₃), 2.58-1.48 (br ov m, 21H). ¹³C{¹H} δ: 160.6, 160.5, 150.7, 138.6, 128.2, 125.0, 72.7, 33.1, 26.4, 26.2, 25.5, 25.4, 24.5. FT-IR: 2924 (m), 2867 (m), 2845 (m), 1645 (m, ν_{CN}), 1596 (m), 1571 (m), 1463 (m), 1395 (m), 1372 (w), 1354 (m), 1340 (m), 1272 (s), 1257 (s), 1220 (s), 1135 (m), 1099 (m), 1069 (w), 1028 (m), 1009 (w), 966 (s), 945 (w), 918 (w), 868 (w), 844 (s), 799 (m), 765 (m), 741 (s), 644 (s).

5.4.5 Synthesis of compound **2e**



A CH₂Cl₂ (3 mL) solution of **1e** (55 mg, 0.27 mmol) was added to [PtCl₂(coe)]₂ (100 mg, 0.13 mmol) in CH₂Cl₂ (5 mL). The reaction mixture was stirred for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield a bright yellow solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 135 mg (23%); m.p.: 135-139 °C (decomp.). NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.84 (s, *J*_{HPt} = 18 Hz, 1H, *H*C=N), 7.83 (t, *J*_{HH} = 8 Hz, 1H, Ar), 7.51 (ov d, *J*_{HH} = 8 Hz, 1H, Ar), 3.94 (br d, *J*_{HPt} = 35 Hz, *J*_{HH} = 8 Hz, 2H), 3.25 (s, 3H, CH₃), 2.18-1.39 (br ov m, 23H). ¹³C{¹H} δ: 160.5, 160.1, 150.7, 138.6, 128.2, 125.0, 71.3, 33.8, 26.4, 26.2, 25.5, 25.2, 25.1. FT-IR: 2922 (m), 2856 (m), 1646 (m, *ν*_{CN}), 1597 (m), 1570 (m), 1463 (m), 1445 (m), 1390 (m), 1372 (w), 1353 (m), 1256 (s), 1162 (w), 1098 (s), 1077 (s), 1028 (s), 964 (s), 947 (s), 924 (s), 887 (s), 866 (w), 799 (m), 673 (s). *Anal.* calc. for C₂₁H₃₁N₂Cl₂Pt • CH₂Cl₂ (577.49 g/mol) (%): C 39.55, H 5.17, N 4.18; found: C 39.89, H 5.02, N 4.23.

5.4.6 Synthesis of compound **2f**



A CH₂Cl₂ (3 mL) solution of **1f** (59 mg, 0.31 mmol) was added to [PtCl₂(coe)]₂ (100 mg, 0.13 mmol) in CH₂Cl₂ (5 mL). The reaction mixture was stirred for 18h at RT under atmospheric nitrogen. Upon removal from the glovebox, the solvent was removed under vacuum to yield an orange solid. The resulting product was washed with hexane (10 mL) and stirred overnight. Yield: 104 mg (64%); m.p.: 173.1-174.8 °C (decomp.). NMR spectroscopic data (CDCl₃, ppm): ¹H δ: 8.80 (s, *J*_{HPt} = 17 Hz, 1H, HC=N), 7.83 (t, *J*_{HH} = 8 Hz, 1H, Ar), 7.54 (ov d, *J*_{HH} = 8 Hz, 1H, Ar), 3.94 (br d, *J*_{HPt} = 41 Hz, *J*_{HH} = 11 Hz, 2H), 3.25 (s, 3H, CH₃), 2.18-1.39 (br ov m, 25H). ¹³C{¹H} δ: 160.5, 159.5, 150.9, 138.6, 131.0, 128.2, 125.0, 73.2, 33.6, 26.4, 26.5, 25.7, 25.4, 24.7, 24.2. FT-IR: 2918 (m), 2852 (m), 1645 (m, ν_{CN}), 1597 (s), 1571 (s), 1463 (m), 1445 (m), 1378 (m), 1358 (m), 1278 (s), 1255 (s), 1217 (s), 1161 (s), 1136 (s), 1095 (s), 1063 (s), 1033 (s), 966 (s), 863 (w), 811 (m), 791 (m), 740 (s), 702 (s), 663 (s). *Anal.* calc. for C₂₃H₃₅N₂Cl₂Pt • CH₂Cl₂ (605.54 g/mol) (%): C 41.15, H 5.55, N 4.72; found: C 41.75, H 5.40, N 4.06.

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Appendix A: Selected Spectra

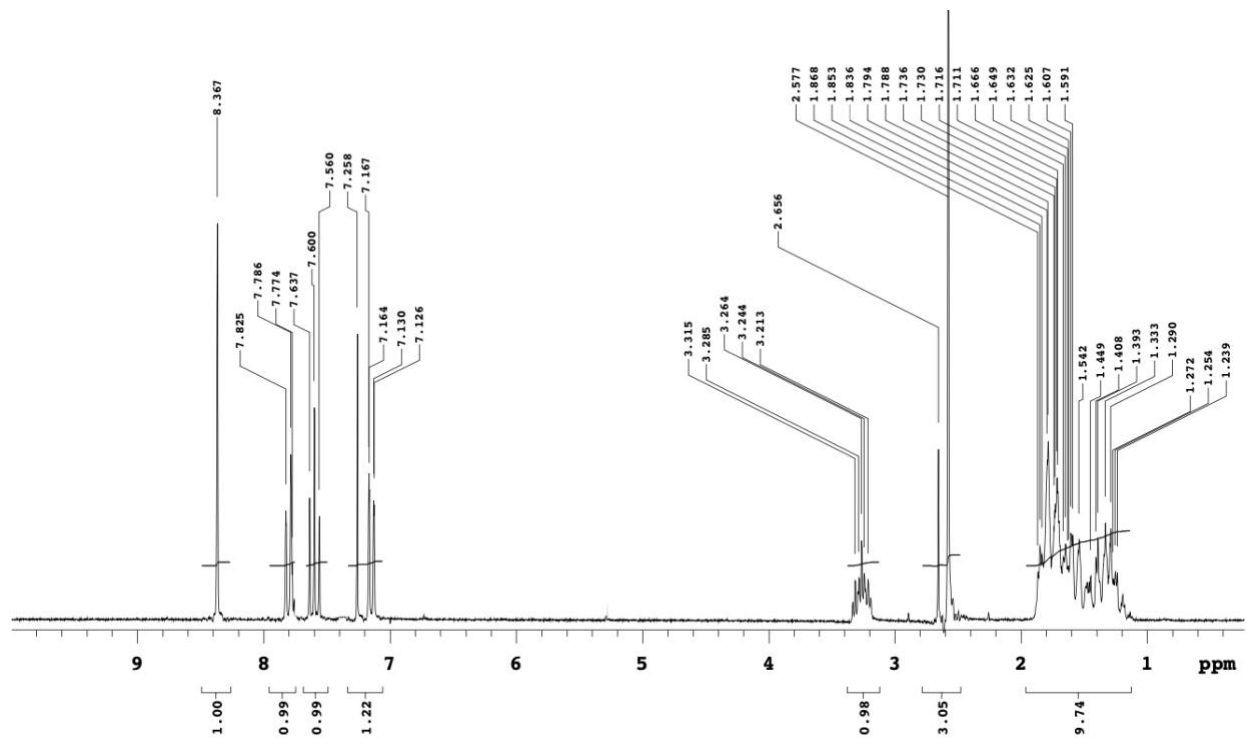


Figure A1. ^1H NMR spectrum of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclohexanamine (**1e**) in CDCl_3 .

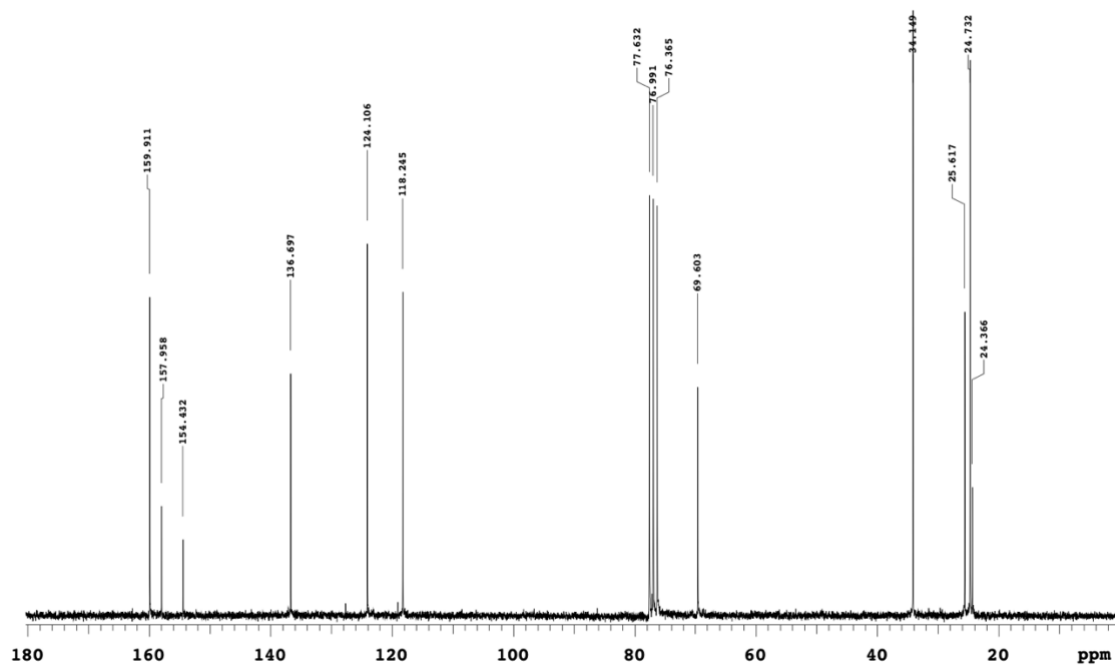


Figure A2. ^{13}C $\{^1\text{H}\}$ NMR spectrum of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclohexanamine (**1e**) in CDCl_3 .

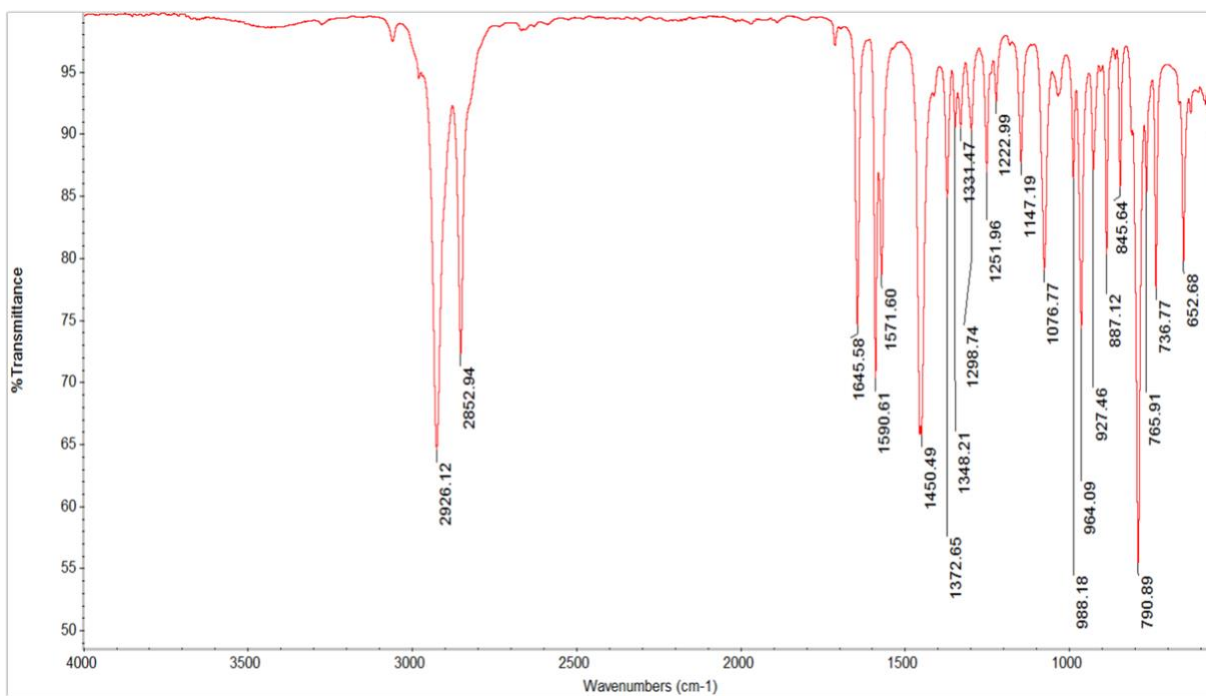


Figure A3. FT-IR spectrum of (Z)-N-((6-methylpyridin-2-yl)methylene)cyclohexanamine (**1e**).

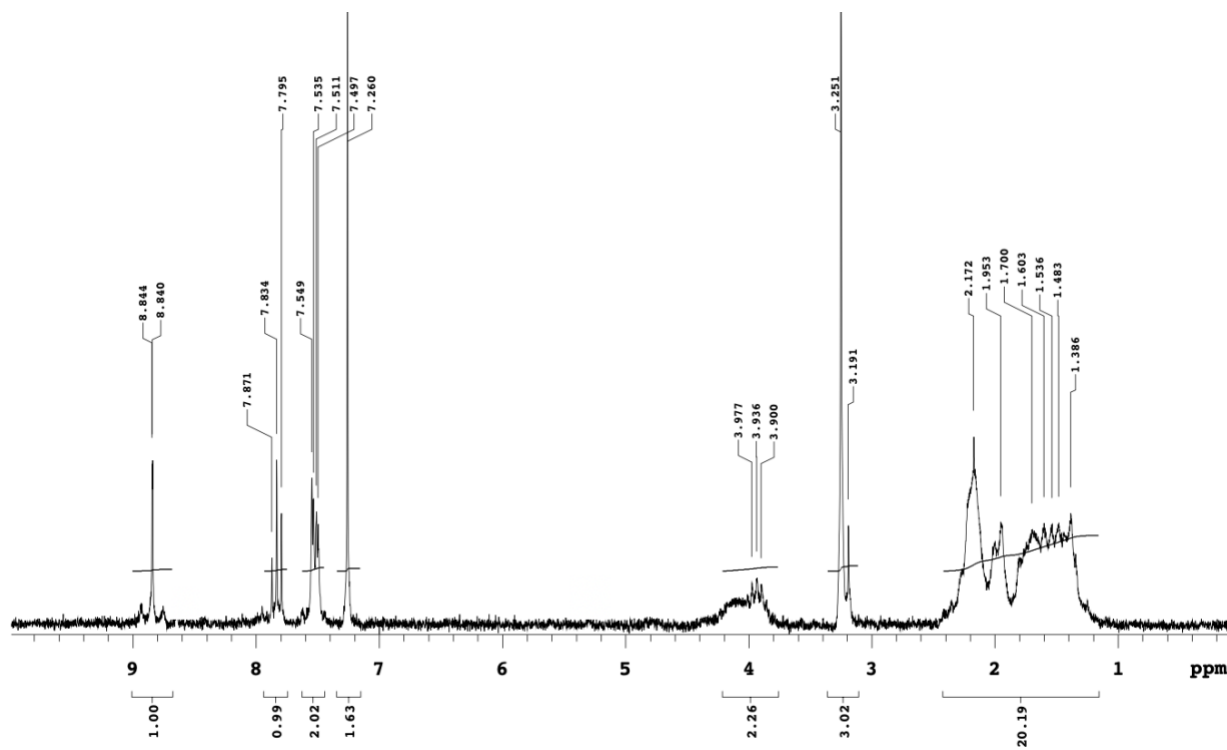


Figure A4. ¹H NMR spectrum of compound **2e** in CDCl₃.

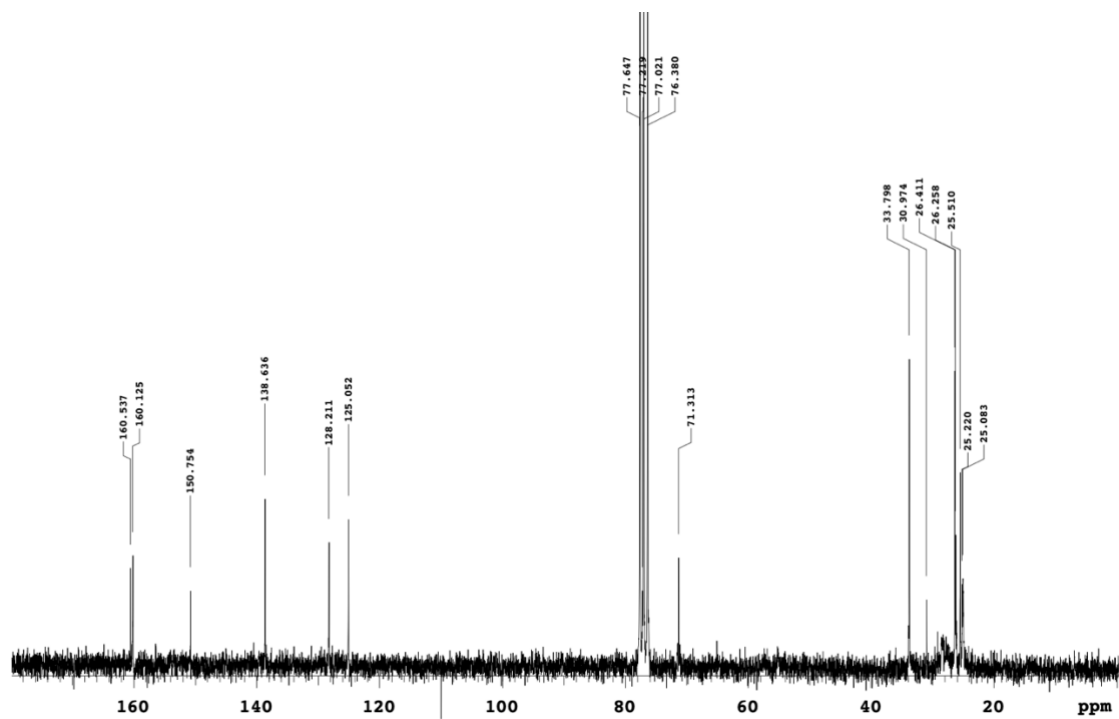


Figure A5. ^{13}C $\{^1\text{H}\}$ NMR spectrum of compound **2e** in CDCl_3 .

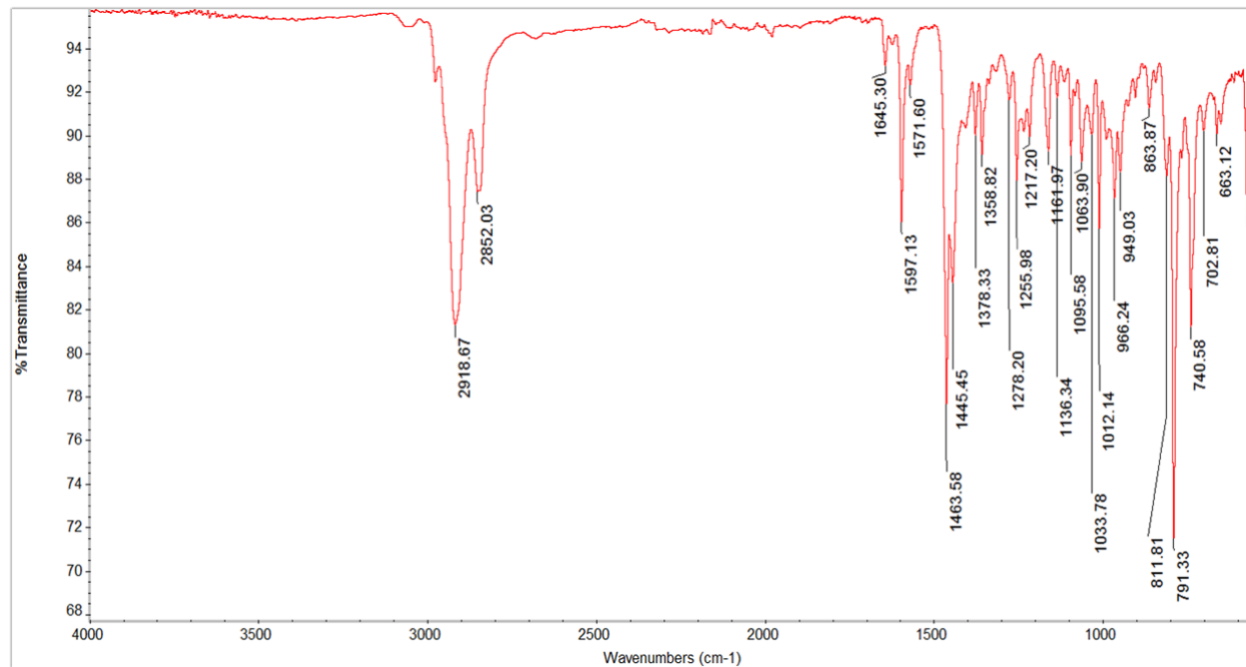


Figure A6. FT-IR spectrum of compound **2e**.

Appendix B: Curriculum Vitae

Khyati Mittal

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Education

Mount Allison University, Sackville, NB Bachelor of Science, Honors Biochemistry	(2019-2022)
York University, Toronto, ON Integrated Science	(2018-2019)
St. Augustine Secondary school, Brampton, ON High School Diploma	(2015-2018)

Awards

R.P. Chapman Award	(2021)
Jeff 'Skip' Fraser Memorial Scholarship	(2021)
Mount Allison Dean's List	(2019-2021)
Mount Allison University Scholarship	(2019-2021)
York University Entrance Scholarship	(2018-2019)
Co-curricular Award	(2017)

Research Experience

Honors Research Thesis: Wild Toads Laboratory (May 2021-)

Mount Allison University, Sackville, NB

- Worked with Dr. Stephen Westcott to synthesize novel platinum(II) complexes containing bulky amine groups for potential anti-cancer activity. These compounds were characterized using multinuclear NMR analysis, melting point assessment, elemental analysis, and FT-IR spectroscopy.

Research Assistant: RadScholars Inc. (Jan 2021-Feb 2022)

- Worked on a systematic review that examines the application of sonography in conducting prenatal autopsies for the diagnosis of trisomy disorders.

Research Assistant: Wild Toads Lab (Jan 2021-Apr 2021)

Mount Allison University, Sackville, NB

- Conducted experimental research in Dr. S. Westcott's laboratory investigating the synthesis of novel iron(III) complexes as potential green pesticides.

Conferences

‘Synthesis and Characterization of Novel Platinum(II) Complexes Containing Bulky Amine Groups’ **K. Mittal**. Summer Undergraduate Research Fair, Sackville, NB, September 2021.

‘Synthesis and Characterization of tris(3-hydroxy-4- pyridinonate)iron(III) complex as a potential green pesticide’ **K. Mittal**. Western Student Research Conference 2021, London, ON, March 2021.

Work Experience

Peer Tutor (Dec 2020-Apr 2021)
Mount Allison University, Sackville, NB
Worked as an academic tutor to assist students who require peer support in courses such as biochemistry, chemistry, biology, and psychology.

Student Teaching Assistant (Feb 2020-Dec 2021)
Mount Allison University, Sackville, NB
Supervised laboratory exercises and demonstrated common lab techniques in Introductory Chemistry II and Inorganic Chemistry I.

Volunteer Experience

Influenza Vaccine Clinic Volunteer (Oct 2021)
Mount Allison University, Sackville, NB
Assist healthcare professionals during the vaccine clinic, ensure line-ups are moving smoothly, sanitize stations, and provide directions.

COVID-19 Vaccine Clinic Volunteer (May 2021-Aug 2021)
Moncton, NB
Responsible for tasks such as monitoring the post-immunization waiting rooms, assisting people with consent forms, ensuring that patients are following proper COVID-19 guidelines, as well as promoting a safe-environment for both the patients and the clinical staff.

Science Atlantic Student Chemistry Conference (May 2021)
Mount Allison University
Hosted the “Non-academic career path” panel discussion in the ChemCon 2021 held at the MTA University, which involved engaging and discussing with acclaimed professionals regarding their non-academic careers.

Friendly Call Program Volunteer (Feb 2021-Nov 2021)
Canadian Red Cross
Responsible for engaging with senior clients as well as promoting local community events, programs, and encourage their participation.

SMILE program mentor (Sept 2020-Dec 2020)
Amherst, NS
Participated in weekly activities with children and teenagers with intellectual and developmental disabilities

Friendly Visitor (Jan 2020-Dec 2021)
Sackville Memorial Hospital, NB
Provide support to senior citizens by keeping them company and engaging them in conversation.

Front-Desk Volunteer (Dec 2019-Apr 2020)
Cumberland Regional Health Care Centre, NS
Positioned as a Way-finder volunteer to provide directions, assist patients and alleviate some stress for patients, visitors, and staff finding locations within the healthcare facility.

Leadership

President, Shotokan Karate Club (Jul 2021-)
Mount Allison University, Sackville, NB
Responsible to book training space and serve as a liaison between the campus and the karate organization.

President, Champions of Change Club (May 2021-)
Mount Allison University, Sackville, NB
Responsible for holding meetings, assist with fundraising and outreach activities, delegate tasks and responsibilities, coordinate the main club events, and serve as a liaison between the club and the campus.

Biochemistry Discipline Representative, Women in Science (May 2021-)
Mount Allison University, Sackville, NB
Responsible for posting weekly posts and features that aim to advocate for rights of those who identify as a woman, as well as encourage and empower those women who are associated with the field of science.

General Fundraising Chair, Global Brigades (Apr 2021-Feb 2022)
Mount Allison University, Sackville, NB
Organize several fundraising events and activities over the semester, plan funding, find sponsors, and promote events on social media.

Logistics Captain, Relay for Life Committee (Mar 2021-)
Mount Allison University, Sackville, NB
Responsible for meeting minutes, coordinate and organize the Relay for Life event and recruit team members.

References

Dr. Stephen Westcott, Professor, Tier I Canada Research Chair, Mount Allison University, Sackville NB, 1-506-364-2372, swestcott@mta.ca

Dr. Tyson MacCormack, Associate Professor, Mount Allison University, Sackville NB, 1-506-364-2371, tmaccormack@mta.ca