

TARGETING CANCER THROUGH THE INHIBITION OF DNA UNZIPPING

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INTRODUCTION

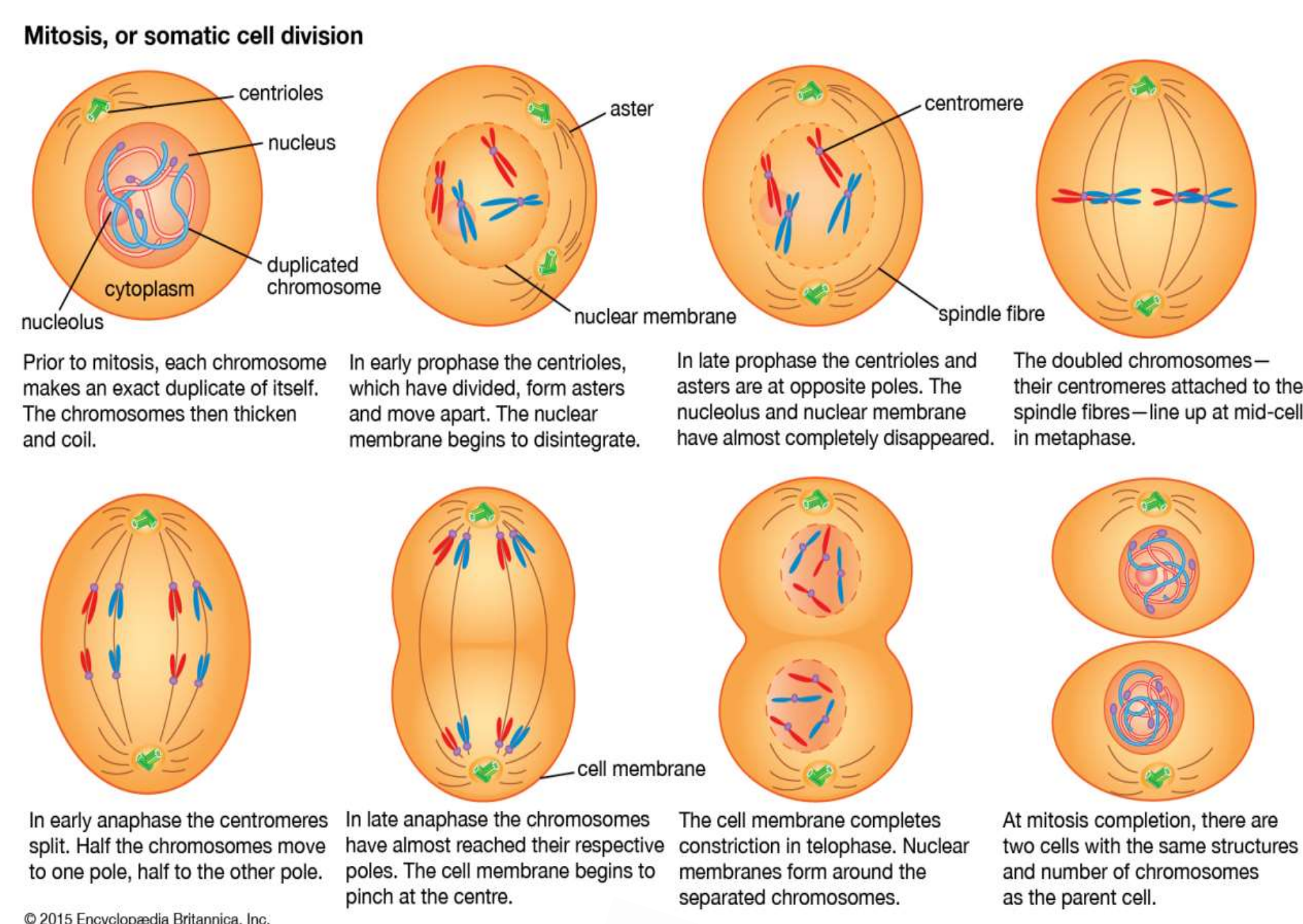


Fig 1. Cancer Invasion and division

AIM OF THE STUDY

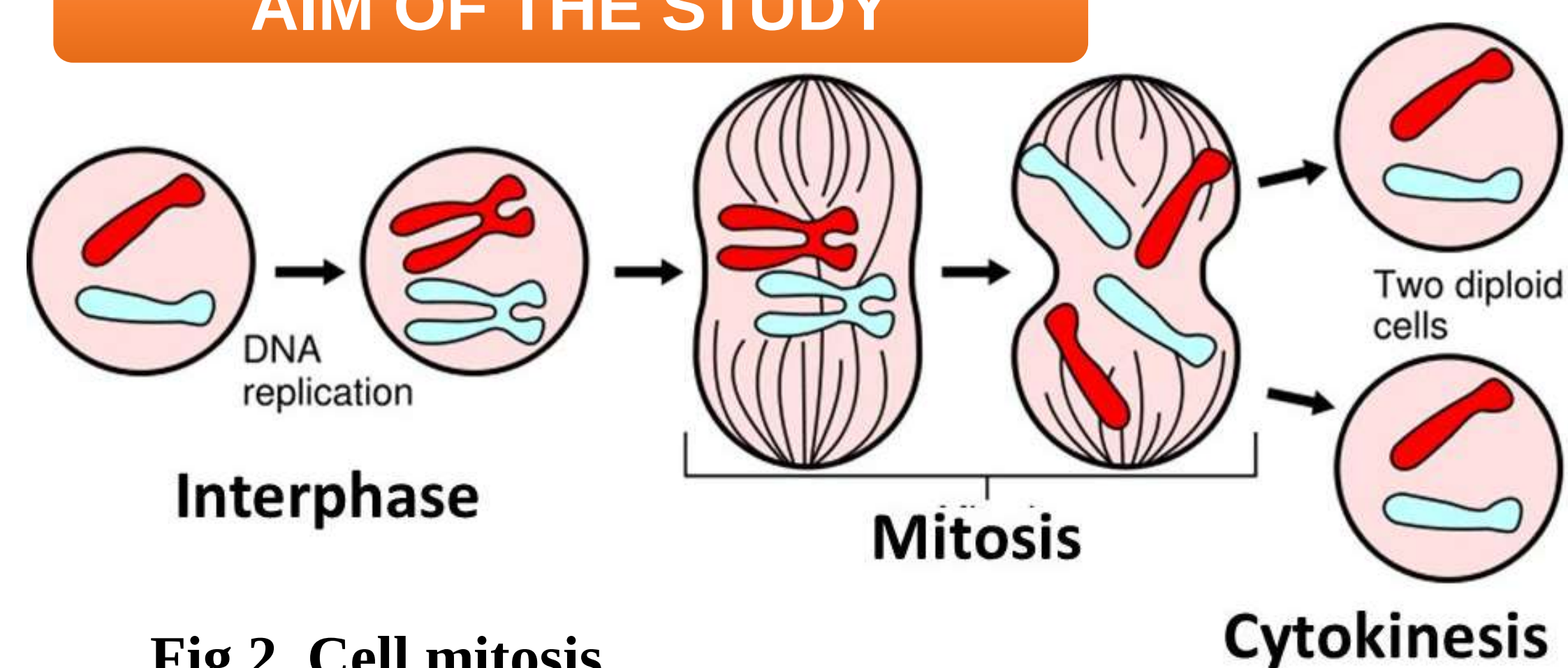
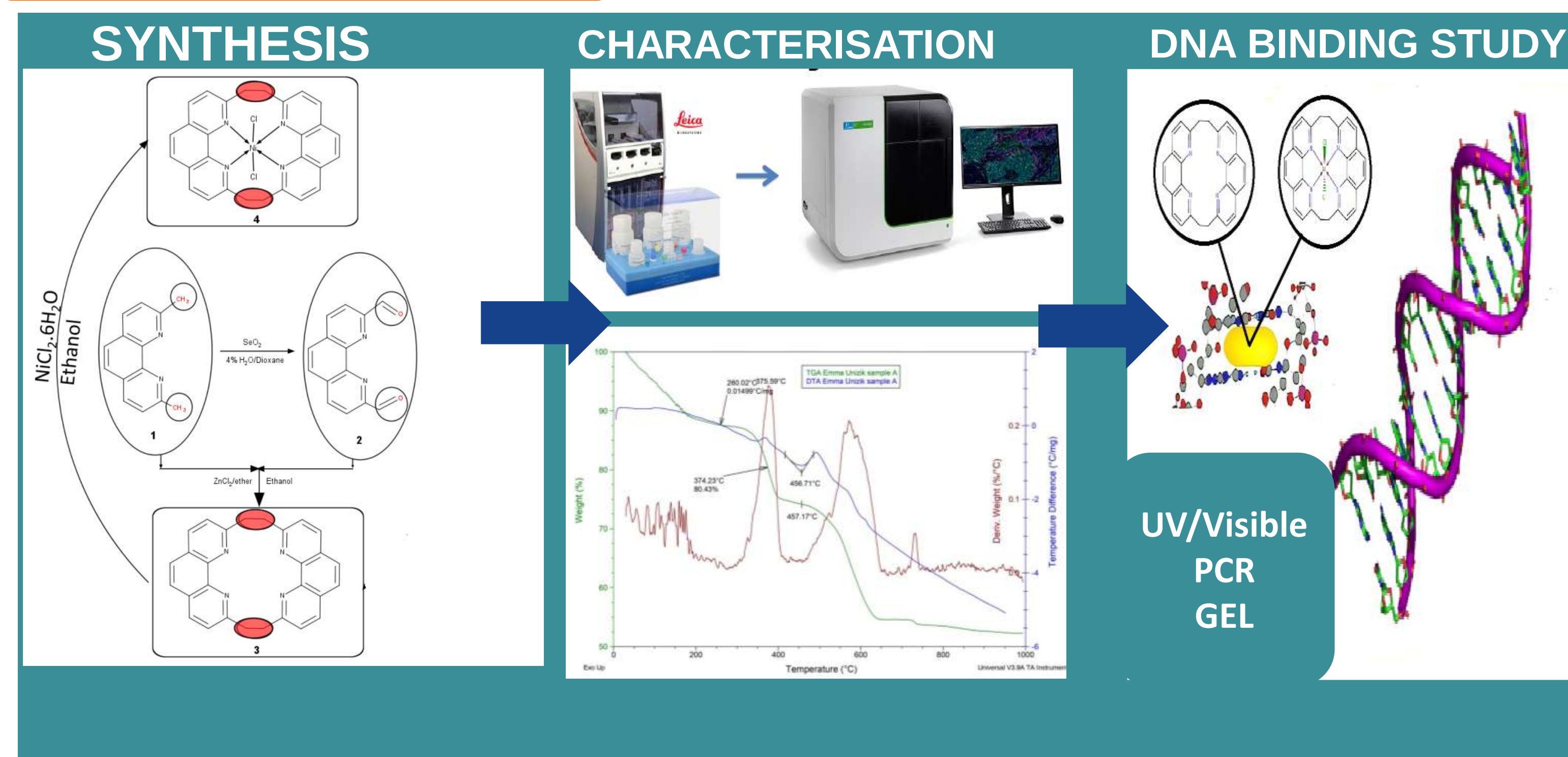


Fig 2. Cell mitosis

Fig 3: DNA-Drug Interaction

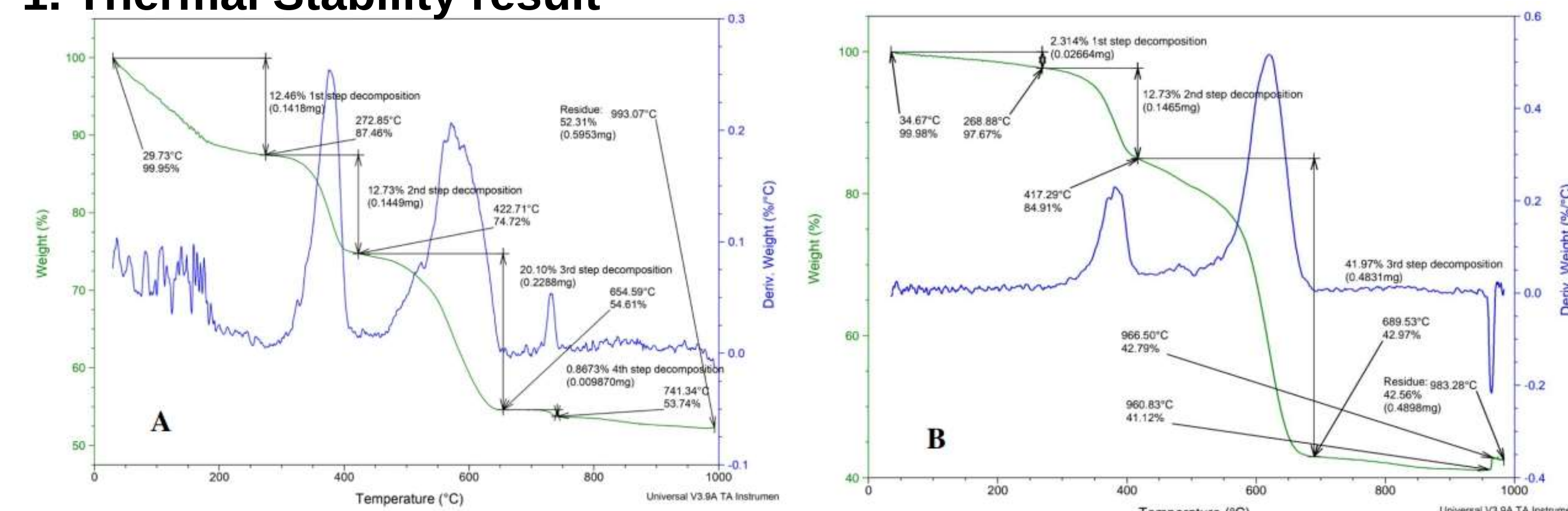


METHODOLOGY



RESULTS AND DISCUSSIONS

1. Thermal Stability result



2. Biophysical characterization of The Drug-DNA interaction

Table 1: Biophysical parameters of the drug interactions with DNA and Amplicon

Samples	Kb/M	ΔG kJ/mol	RMSD(Å)	Fsum of Sqr	Degree of Freedom
SampleA_DNA	1.00×10^6	-35.6	0.71	2.54	5
SampleB_DNA	1.07×10^8	-47.7	0.56	1.56	5
Sample A_Amplicon	6.00×10^4	-28.4	0.74	2.77	5
Sample B_Amplicon	6.00×10^6	-40.2	0.620	1.92	5

CONCLUSION

- Show reasonable DNA base and GC-DNA amplicon binding affinities of 1.00×10^6 M⁻¹ and 6.00×10^4 M⁻¹ for the ligand and 1.07×10^8 M⁻¹ and 6.02×10^6 M⁻¹ for the Ni-ligand complex and can therefore, potentially interfere with DNA replication and repair processes.
- Have UV-Visible spectroscopic properties that point to non-covalent binding of the electrostatic, intercalative and groove binding types for the DNA types (dsDNA and GC-DNA amplicon).
- Demonstrated DNA intercalation, groove and electrostatic binding capable of limiting DNA PCR amplification.

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ACKNOWLEDGEMENT

This research was supported by the Academic Staff Union of Universities (ASUU). We acknowledge the support from Molecular Research Foundation for Students and Scientists and the Federation of Commonwealth Chemical Sciences Societies for this opportunity provided for one of us (ECO) to attend this conference and present our research.