Challenges in the Design of Coordination Polymers of the Zinc-Triad 1,1-Dithiolates

Edward R.T. Tiekink

Research Centre for Crystalline Materials
Faculty of Science and Technology

International Conference On Mathematics And Natural Sciences 2017

Why molecules pack as they do?

1,1-dithiolates

Secondary bonding

Extended architectures mediated by bipyridyl bridges

Perplexing results

Intermolecular interactions involving chelate rings

Why molecules pack as they do?

1,1-dithiolates

Biological bismuth

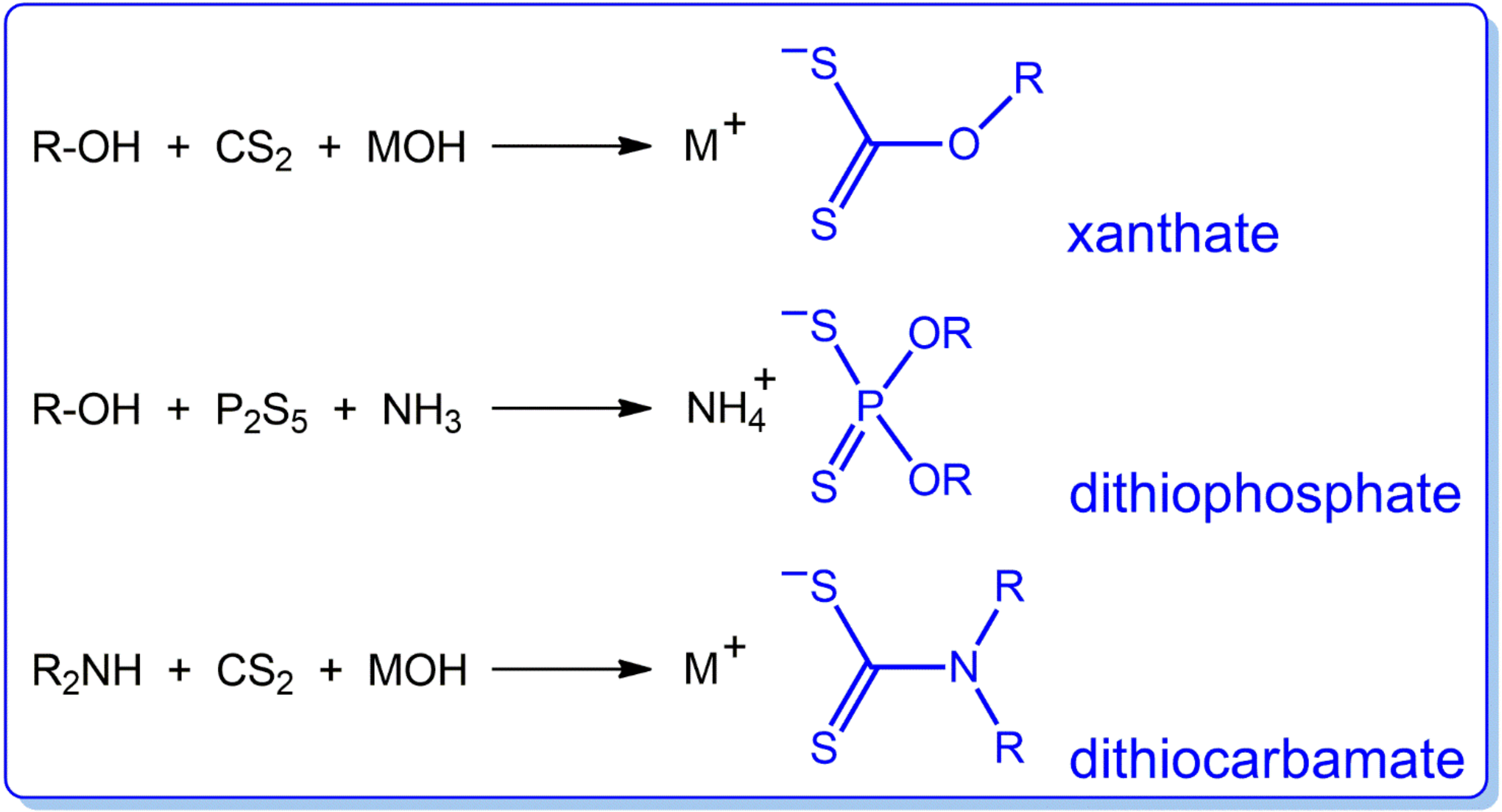
Secondary bonding

Extended architectures mediated by bipyridyl bridges

Perplexing results

Intermolecular interactions involving chelate rings

Synthesis

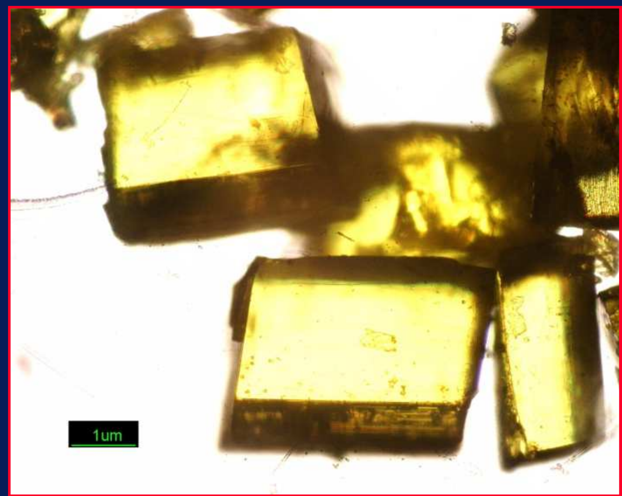


Metathesis: $AX_2 + 2\ MS_2COR \longrightarrow A(S_2COR)_2 + 2MX$

Molecular bismuth compounds, a panacea for human disease?

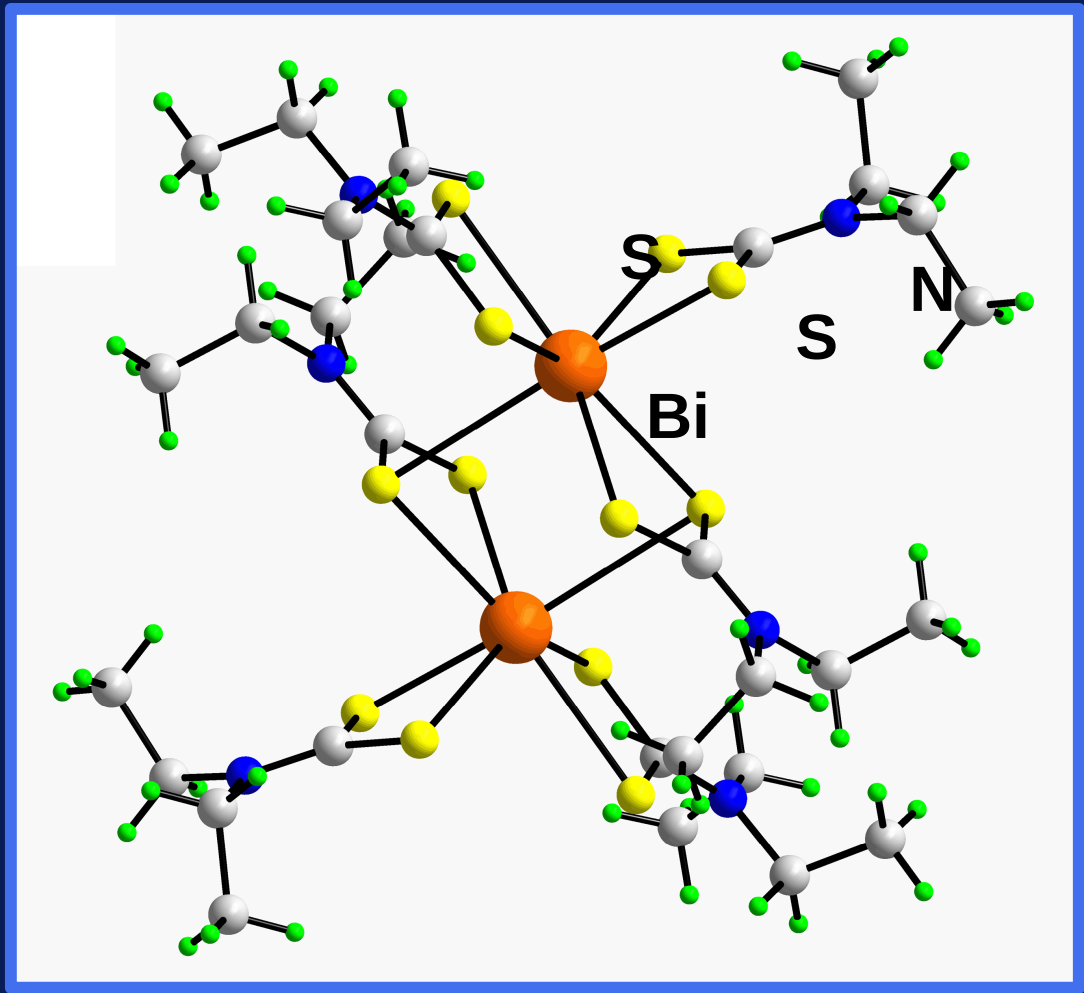
Biological bismuth

Preparation of $\{Bi(S_2CNEt_2)_3\}_2$



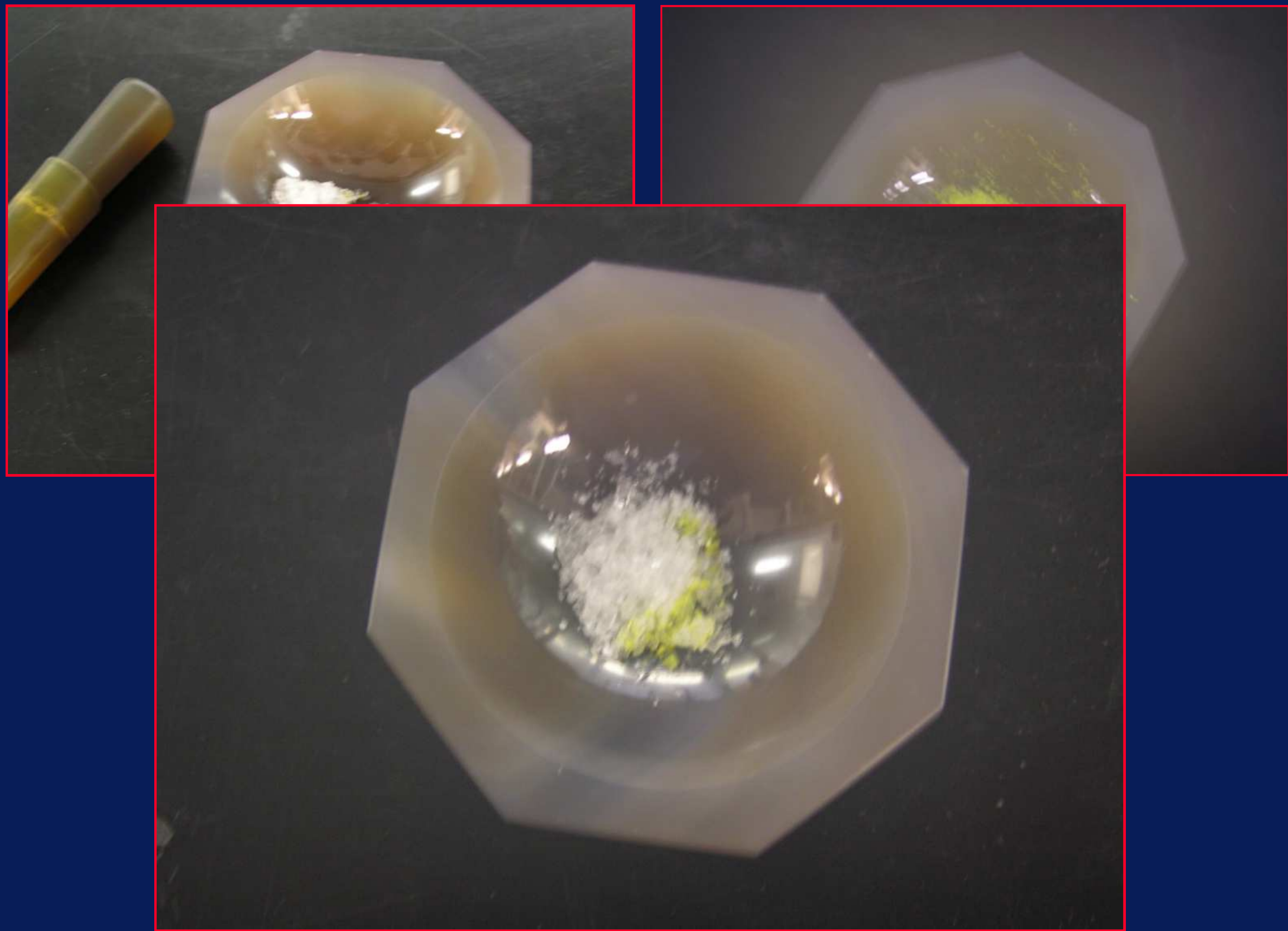
Two hours

Dimeric structure of $\{Bi(S_2CNEt_2)_3\}_2$



Single-crystal X-ray crystallography

Preparation of $\{Bi(S_2CNEt_2)_3\}_2$



Green chemistry

Bismuth and Medicine

gastric and duodenal ulcers:

De-Nol®
(coll-Bi subcitrate)

Pepto-Bismol®
(Bi subsalicylate)

Pylorid®
(rantidine Bi citrate)

262 or 524 mg/ml

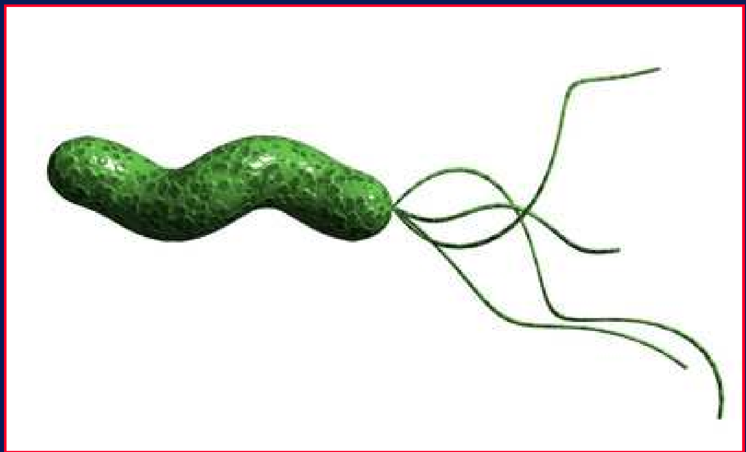
...caused by stress, spicy foods and too much acid

Helicobacter pylori

Harms stomach lining

Stimulates immune response

Inflammation



http://www.pcsg.org.uk/html/dis_helicobacter.html

C & N News, October 10, 2005

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MEDICAL NOBEL PRIZE

ULCER REVELATION

Two Australians share award for discovery of ulcer-causing bacterium

THE 2005 NOBEL PRIZE IN Physiology or Medicine honors the discovery that ulcers are caused by the bacterium *Helicobacter pylori*—not stress or lifestyle as had been thought. The Nobel Assembly at Karolinska Institute, which awards the prize, describes the discovery as "remarkable and unexpected" and notes that it "challenged prevailing dogmas."

J. Robin Warren, 68, formerly a pathologist at the Royal Perth Hospital in Australia, and Barry J. Marshall, 54, who runs an *H. pylori* research lab at the University of Western Australia, in Nedlands, will share the $1.3 million prize.

The path to the Nobel Prize began when Warren noted that stomach tissue from patient biopsies was often colonized by a small, curved bacterium and that inflammation of the gastric mucosa in these tissue samples always occurred near the bacterium. Marshall collaborated with Warren and eventually succeeded in culturing the bacterium, which was later named *H. pylori*. The researchers determined that the bacterium could be found in most patients with gastric inflammation, duodenal ulcer, or gastric ulcer. On the basis of these results, "they proposed that *H. pylori* is involved in the etiology of these diseases," the Nobel Assembly notes.

Because the stomach's acidity was thought to be too harsh for bacteria to survive, the researchers' hypothesis was met with skepticism. Marshall responded by quaffing a culture of the bacterium and showing in a most direct manner that the organism causes gastritis.

"Thanks to the pioneering discovery by Marshall and Warren, peptic ulcer disease is no longer a chronic, frequently disabling condition, but a disease that can be cured by a short regimen of antibiotics and acid secretion inhibitors," the Nobel Assembly says.—SOPHIE ROVNER

ROB DUNCAN/AGENCE FRANCE PRESSE/NEWSCOM

CHEERS Warren (left) and Marshall celebrate their win.

CULPRIT Discovery of *H. pylori*, shown here in an artist's rendition, and its role in causing ulcers was honored with the Nobel Prize.

COURTESY OF WWW.HPYLORI.COM.AU

NOBEL PRIZE IN PHYSICS

OPTICS DISCOVERIES

Americans, German are honored for theoretical and practical breakthroughs

allowed physicists to examine the stability of nature's constants over

/STEPHAN SAVOIA

OTO/ED ANDRIESKI

er.html

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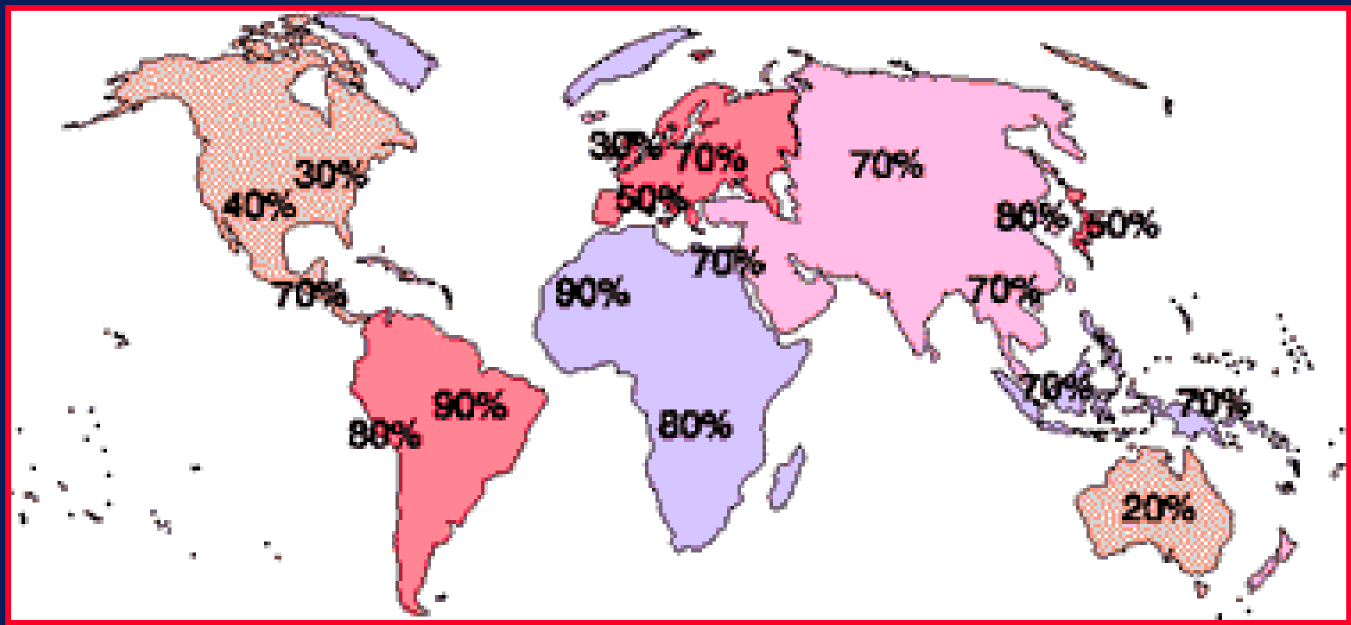
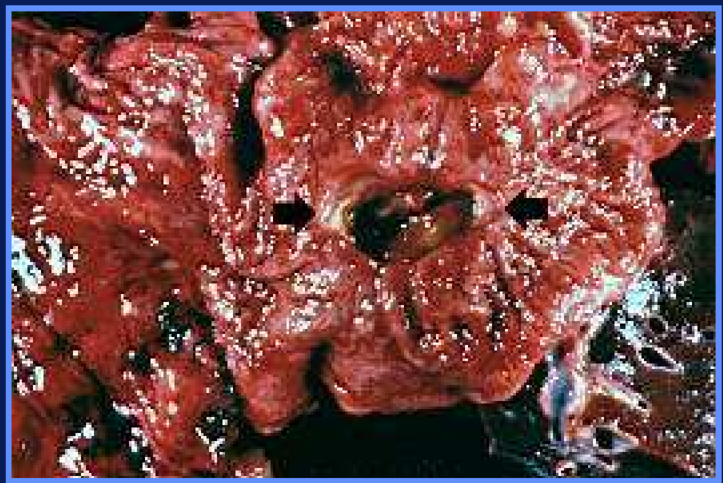
Helicobacter pylori



http://www.helico.com/

Mechanism of action (?):

Helicobacter pylori :



IPNET.LAB
The Interactive Pathology Laboratory

Bismuth subsalicylate

$\downarrow H^+$

"Bi"

Inactivates F1-ATPase

Binding to transferrin and lactroferrin

Bismuth compounds and cancer

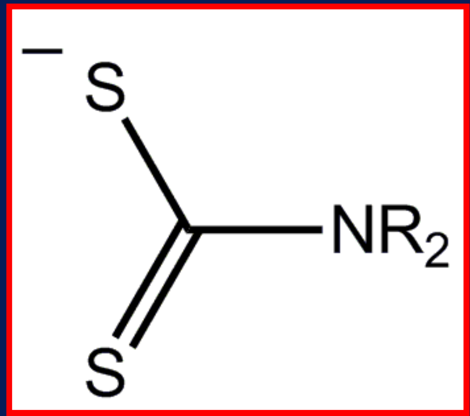
Bismuth compounds: pro-drugs

H.p. causative agent for gastrointestinal cancers

WHO: class 1 carcinogen

Combine bismuth with 'useful' thiols

Dithiocarbamates in biology



Radiation therapy: inhibition of SOD

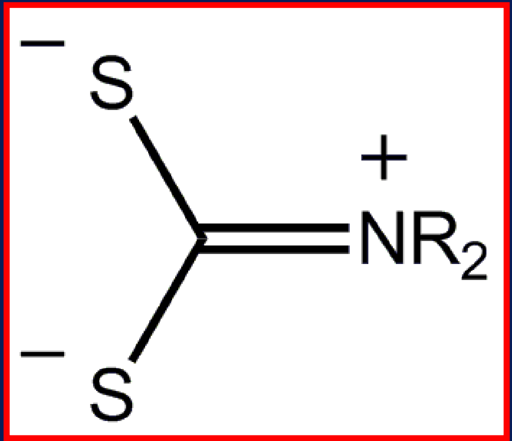
Anti-cancer effects:

reduces alkylation of DNA

combination therapy with cisplatin

Dithiocarbamates in biology

Significant contribution of:



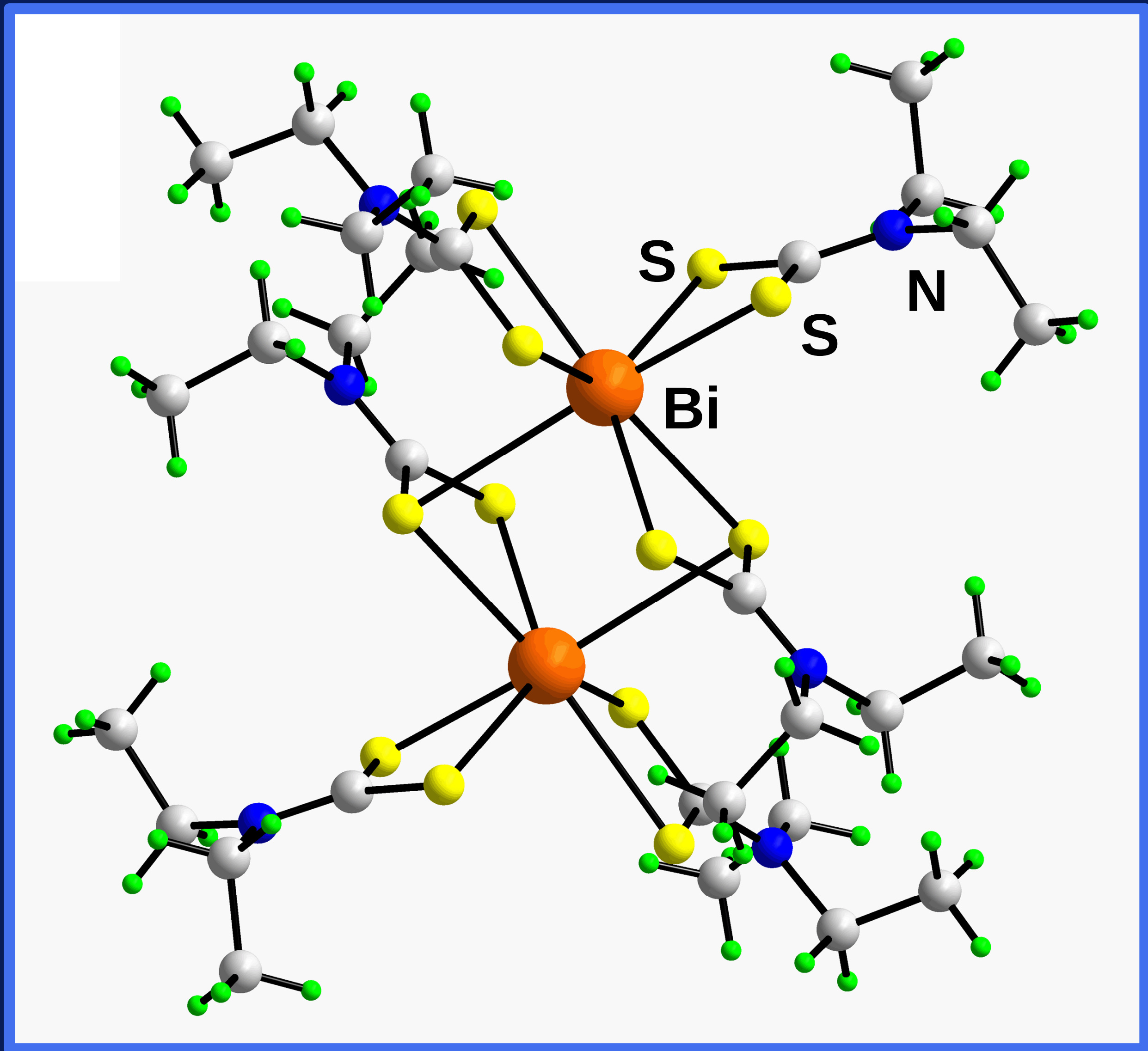
- a very effective chelator for metals

Metal dithiocarbamates in medicine

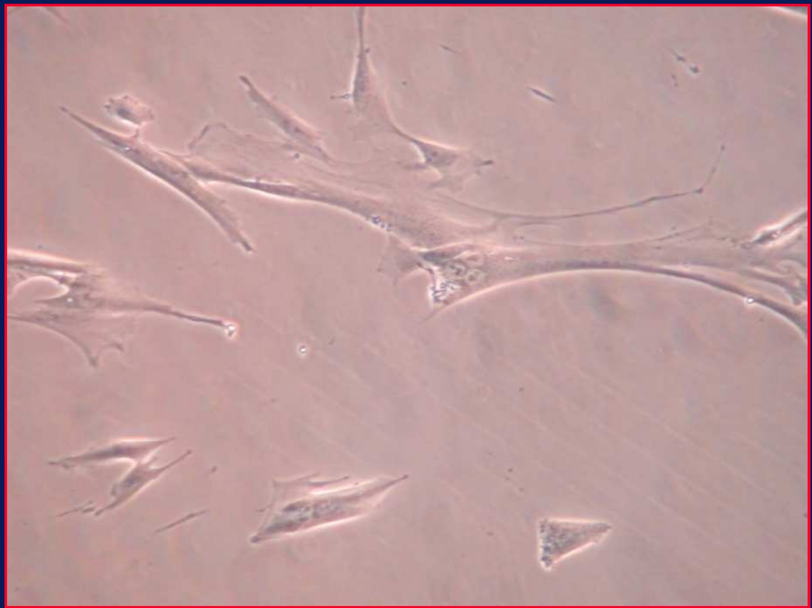
Cu:	Wilson's disease
Ru:	anti-viral
Sn:	anti-microbial
Fe:	anti-HIV
Pt, Pd, Sn & Au:	anti-tumour potential

Hogath (2012)

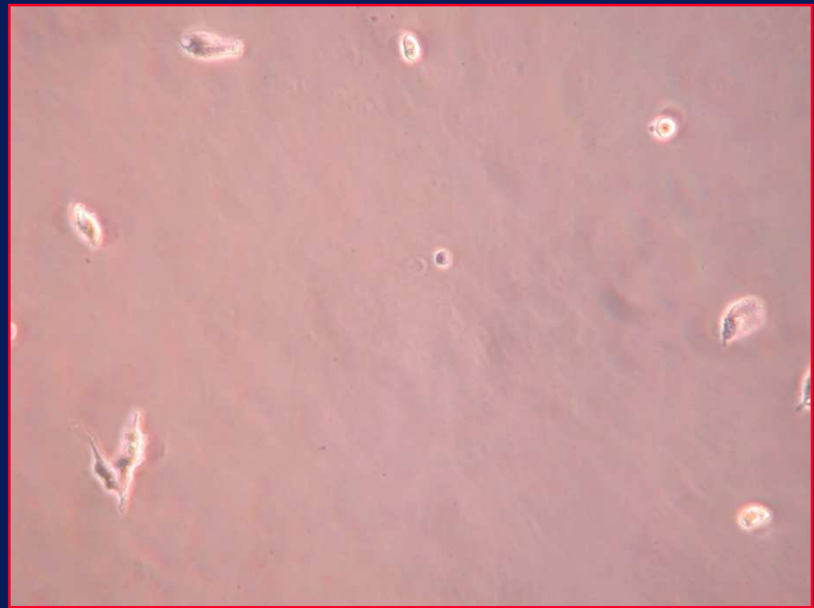
Dimeric structure of $\{Bi(S_2CNEt_2)_3\}_2$



MRC-5 Cells



normal

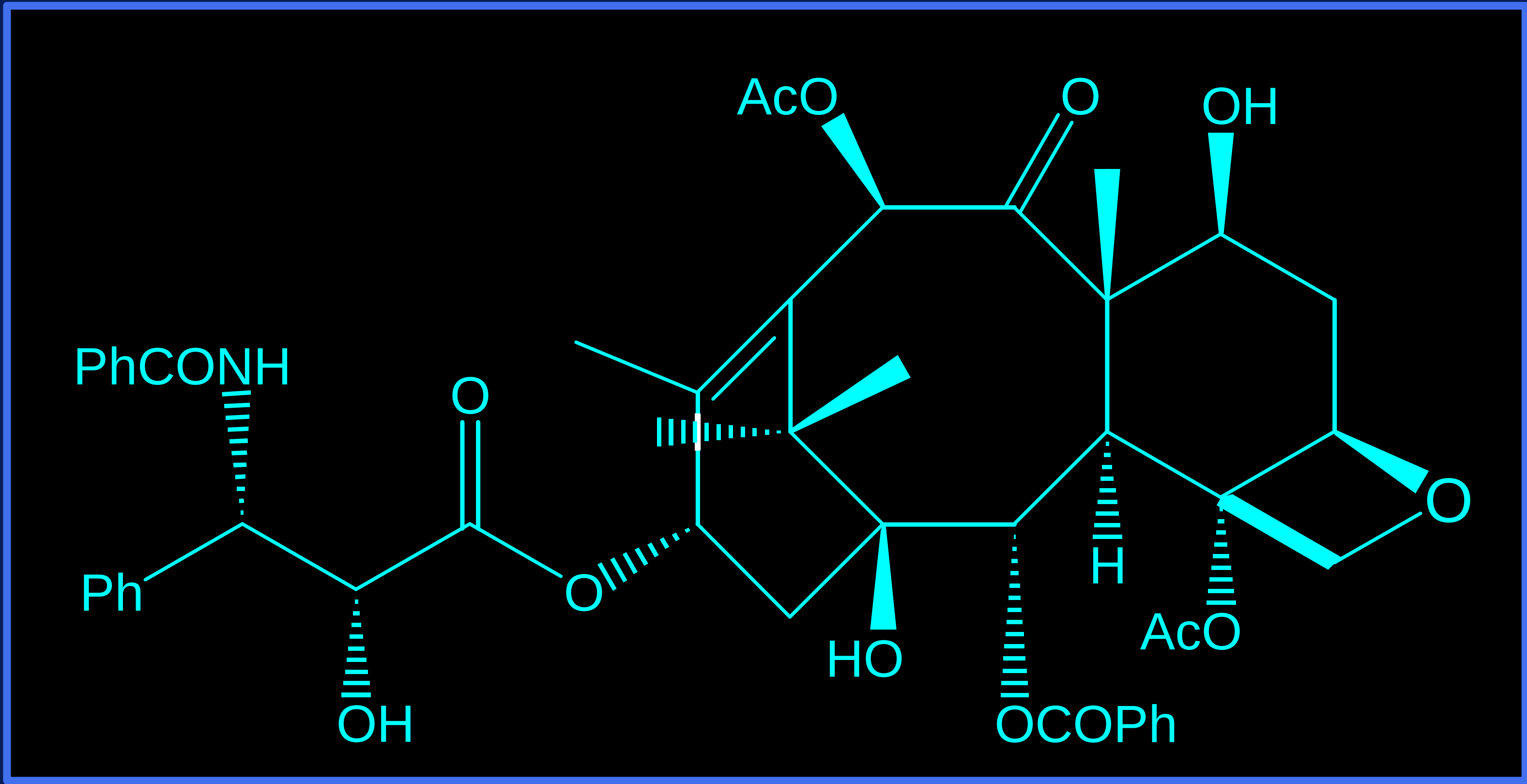


treated

Cytotoxicity (ID_{50}; ng/ml):

	IGROV-1	MCF-7
cisplatin	169	699
DOX	60	10
MTX	7	18
ETO	580	2594
$Bi(dedtc)_3$	< 3.2	4
TAX	< 3.2	< 3.2

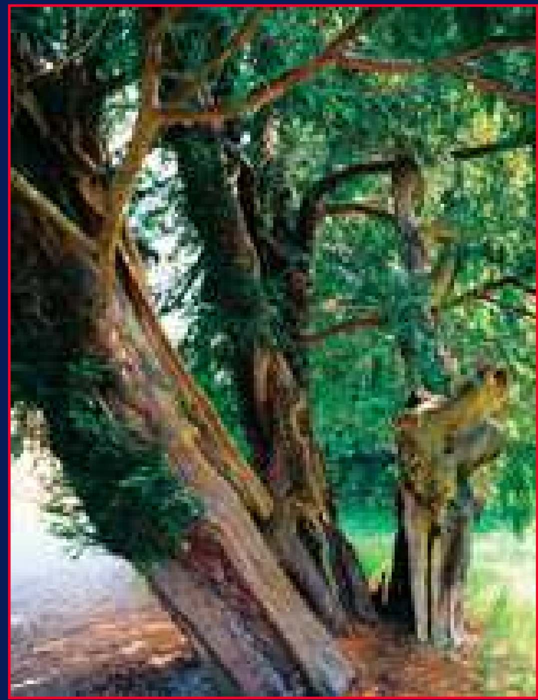
Taxol (Paclitaxel®)



Ovarian & breast cancers, Kaposis' sarcoma, non-small-cell lung cancer

Taxol (Paclitaxel®)

Natural product:
Pacific Yew



Six 100 year old trees / one patient
Ceased harvesting 1993

http://www.research.fsu.edu/researchr/fall2002/taxol.html

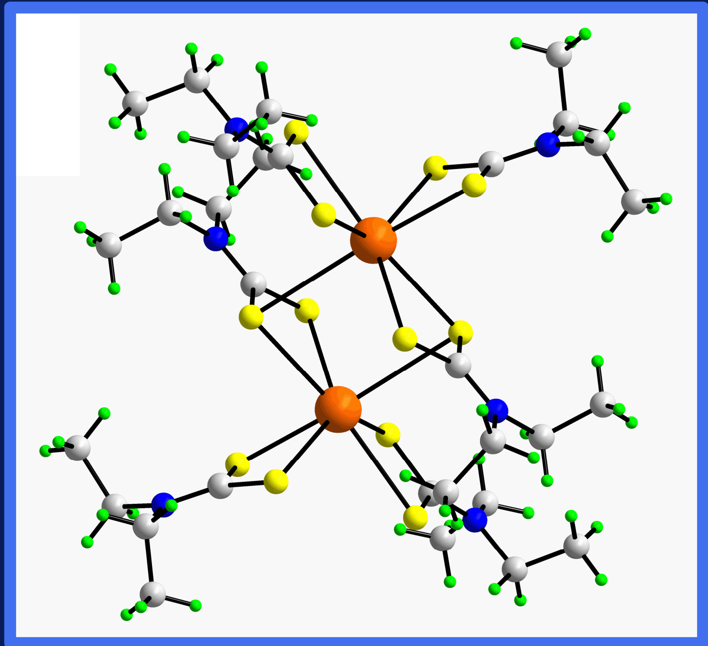
Synthesis of Taxol (Paclitaxel®)

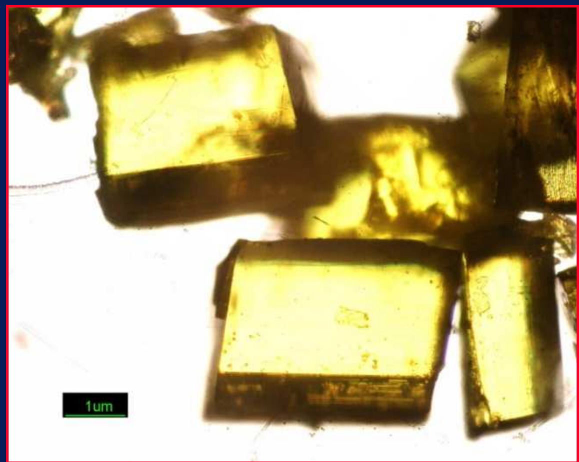
Semi-synthetic route:
taxane from English
yew tree – 35% yield

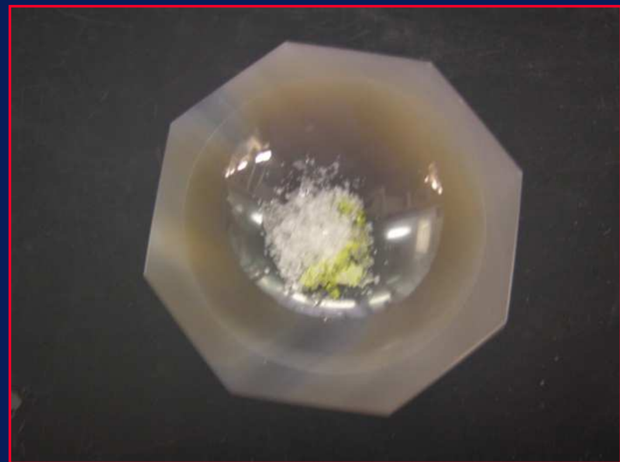
Multi-step organic synthesis
e.g. Holton synthesis: 40 steps – 2% yield

ChemEngNews 2003 Sept. 15th

Preparation of $\{Bi(S_2CNEt_2)_3\}_2$

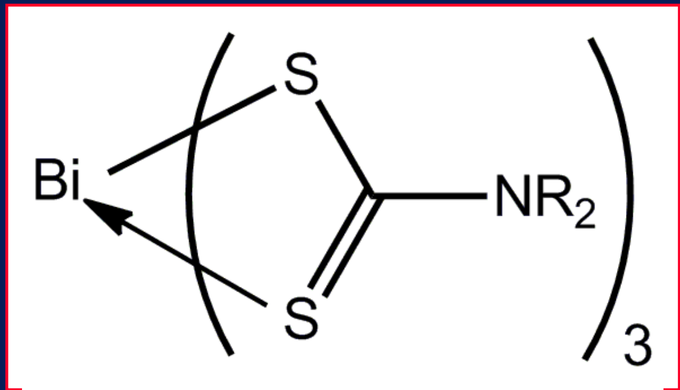






Structure/activity relationship

Alkyl chain	Me < Et > ^{n}Pr > ^{n}Bu
Branching	^{n}Pr > ^{i}Pr & ^{n}Bu > ^{i}Bu
Ring	n = 4 > n = 7
Aromatic	inactive



Maximum Tolerated Dose - Balb/C mice

Four to five weeks old

Male and Female

Controls - 20 day max.

via intraperitoneal injection (i.p.)

Maximum Tolerated Dose – Balb/C mice

$Bi(S_2CNEt_2)_3$:	7 mg Bi / kg
$Bi(S_2CNBu_2)_3$:	> 50 mg Bi / kg

Oral administration

$Bi(S_2CNEt_2)_3$:	~ 50 mg Bi / kg

MTD - Balb/C mice

- Ovcar: Ovarian cancer

MTD = 7 mg Bi / kg

ID_{50} = < 3.2 ng Bi / kg

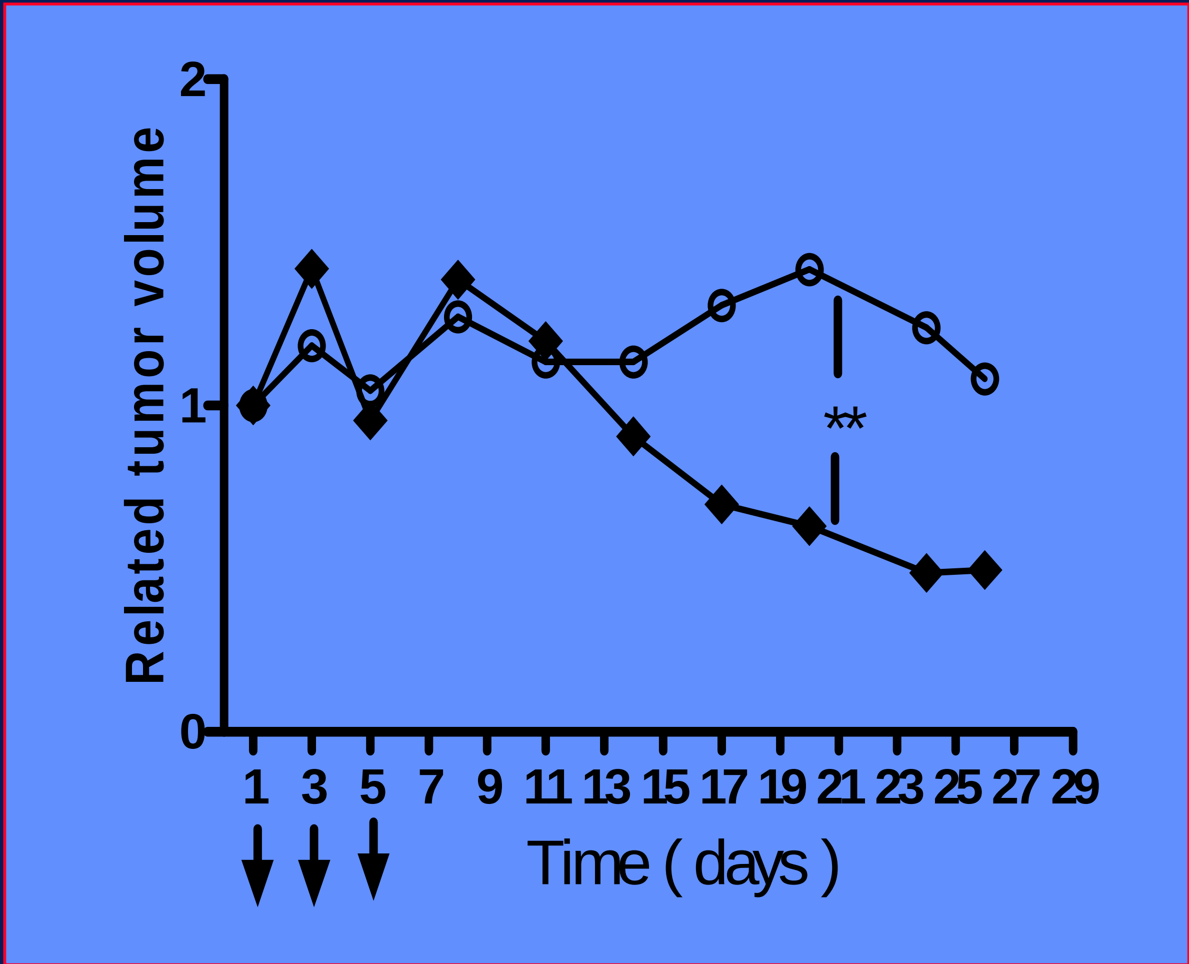
"Therapeutic index"

= > 2.2×10^6

Anti-tumour activity – Balb/C Nude mice

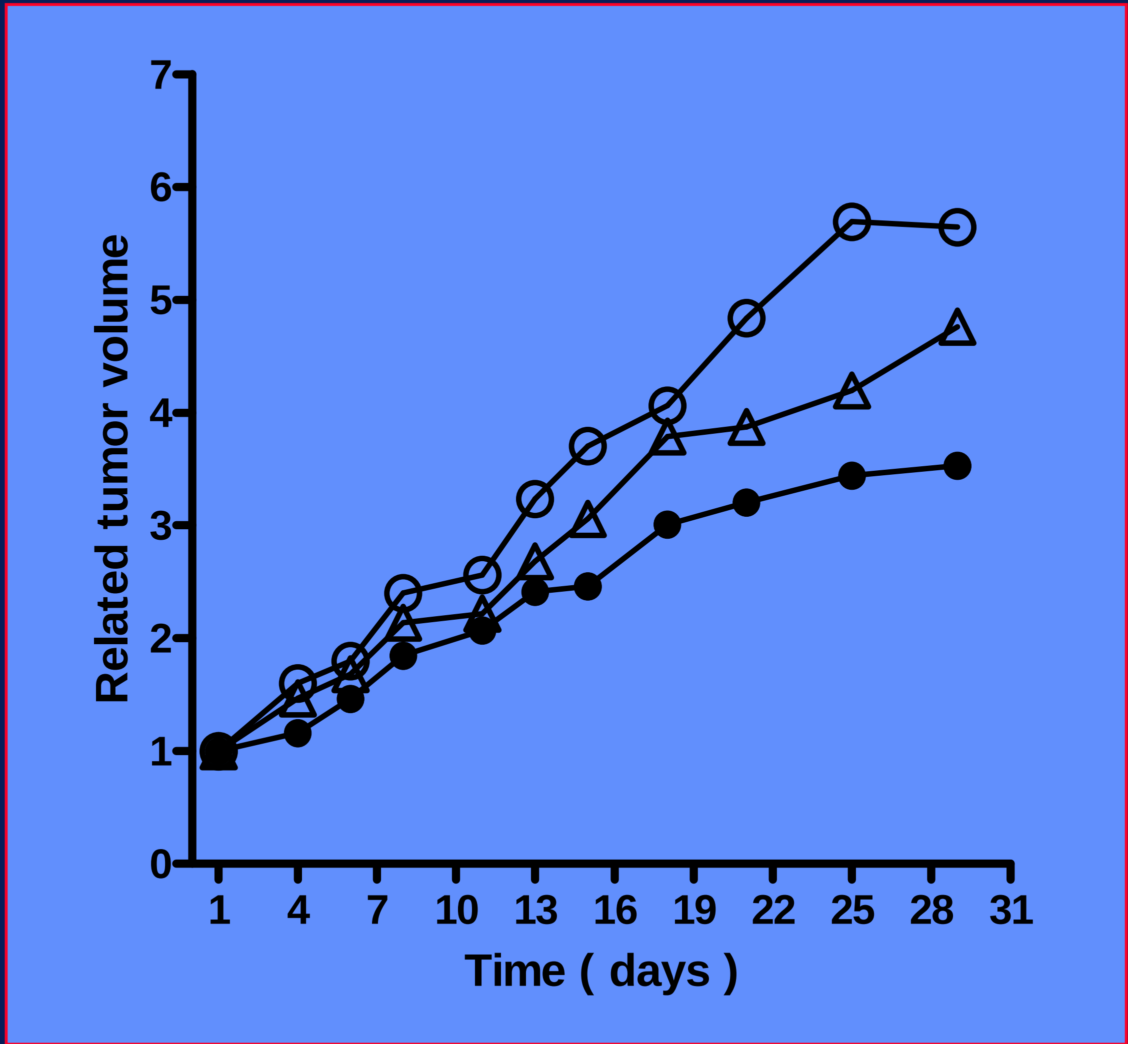
Anti-tumour activity – Balb/C Nude mice

Anti-tumour activity: OVCAR



TWI: 54%
(day 26)

Anti-tumour activity: HT-29



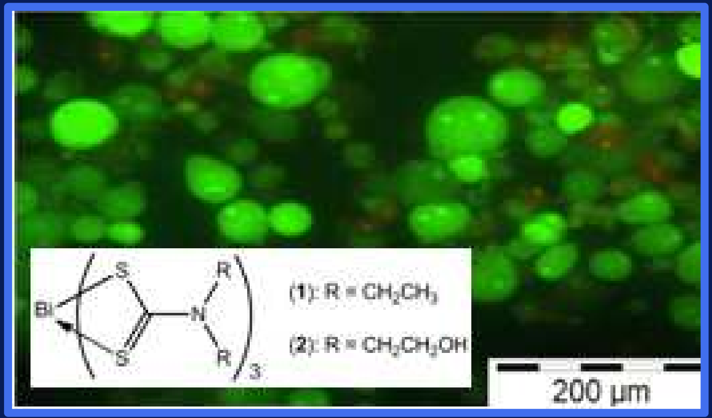
control

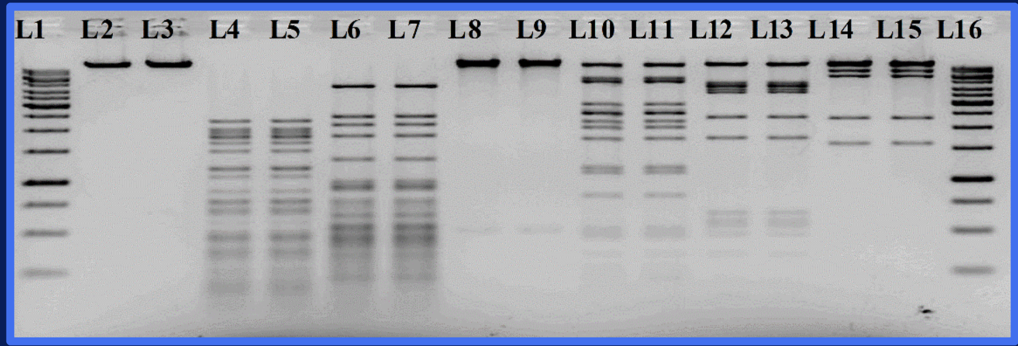
single-dose

multi-dose

(1.4 mg/kg Bi/dose)

HepG2 cell death: apoptosis





Membrane permeability, DNA fragmentation, caspase activities and PCR-array analysis indicate

extrinsic and intrinsic pathways

Table 2
Apoptosis gene expression level in HepG2 cell lines after treatment with 1 and 2 compared to untreated cells.[a]

	Up–down regulation fold regulation (comparing to control group)	
	1	2
ABL1	**58.13**	**253.96**
AKT1	**72.06**	**241.94**
APAF1	**33.17**	**12.62**
BAD	**34.56**	**49.81**
BAG1	*−11.62*	**21.51**
BAG3	*−54.99*	**172.26**
BAG4	*−14.13*	**236.96**
BAK1	**64.50**	**164.11**
BAX	**54.99**	**14.04**
BCL10	**11.64**	**26.51**
BCL2	*−16.63*	*−6.33*
BCL2A1	**9.59**	**18.75**
BCL2L1	*−5.70*	**61.33**
BCL2L10	*−8.41*	**1.01**
BCL2L11	**3.08**	**8.16**
BCL2L2	**35.54**	**123.51**
BCLAF1	**93.78**	**393.03**
BFAR	**1.55**	**2.88**
BNIP3	*−87.96*	*−49.92*
BNIP3L	**55.28**	**22.65**
BRAF	*−22.92*	*−8.18*
NOD1	**22.03**	**99.63**
CARD6	**1.01**	**1.09**
CARD8	**3.10**	**3.50**
CASP1	**44.67**	**54.51**
CASP10	**1.01**	**4.43**
CASP14	**1.01**	**33.32**
CASP2	**58.94**	**304.12**
CASP3	**97.69**	**4.91**
CASP4	**8.64**	**9.31**
CASP5	**3.41**	**3.36**
CASP6	**38.35**	**109.78**
CASP7	**27.69**	**159.62**
CASP8	**37.57**	**14.32**
CASP9	**103.34**	**160.95**
CD40	**12.33**	**1.01**
CD40LG	**8.64**	**1.01**
CFLAR	**28.47**	**46.48**
CIDEA	*−1.19*	*−1.19*
CIDEB	**23.77**	**134.22**
CRADD	**37.04**	**112.87**
DAPK1	**42.54**	**1.01**
DFFA	**103.34**	**59.16**
FADD	**7.42**	**18.23**
FAS	**42.85**	**131.46**
FASLG	**6.03**	**8.39**
GADD45A	**4.26**	**2.62**
HRK	**10.06**	**18.11**
IGF1R	*−212.13*	*−86.91*
LTA	**9.52**	**11.22**
LTBR	**215.45**	**395.76**
MCL1	**36.53**	**69.00**
NOL3	**82.21**	**160.73**
PYCARD	**1.04**	**1.01**
RIPK2	**37.82**	**180.83**
TNF	*−7.94*	*−7.95*
TNFRSF10A	**22.49**	**65.73**
TNFRSF10B	**250.94**	**928.32**
TNFRSF11B	**6.28**	**20.80**
TNFRSF1A	**138.26**	**225.73**
TNFRSF21	**12.65**	**51.57**
TNFRSF25	**1.01**	**93.60**

Table 2 (*continued*)

	Up–down regulation fold regulation (comparing to control group)	
	1	2
CD27	*−1.88*	*−1.32*
TNFRSF9	**1.01**	**1.01**
TNFSF10	**2.95**	**2.03**
CD70	**2.93**	**3.29**
TNFSF8	**7.47**	**5.42**
TP53	**113.08**	**90.63**
TP53BP2	**4.41**	**22.76**
TP73	**18.79**	**8.62**
TRADD	**35.29**	**92.31**
TRAF2	*−1.05*	**77.11**
TRAF3	*−3.14*	**73.46**
TRAF4	*−11.74*	**51.98**

Bold face: up-regulated genes; italic: down-regulated genes.
[a] Data represent mean of samples 1 and 2-induced fold-change in gene expression relative to control-treated cells (n = 3), $p < 0.05$.

receptors of the tumour necrosis factor receptor superfamily, member 6

TP53 113.08 90.63
TP53BP2 4.41 22.76
TP73 18.79 8.62

BCL-2 family member BID, a molecular linker bridging death receptor and mitochondria pathways to truncated BH3 interacting-domain death agonist (tBID), that binds to pro-apoptotic protein BAX, resulting in the disruption of mitochondrial membrane potential and the release of cytochrome c [54–56]. Compounds 1 and 2 also induced expression of BID in HepG2 cells (Table 2), which provides additional supporting evidence for the proposed apoptotic pathways.

Interestingly, apart from the mitochondrial and FAS death receptor apoptotic pathway, 1 was also found to induce another death receptor-dependent pathway by induction of a series of pro-apoptotic genes, such as CD40 (CD40 gene), CD40L (CD40LG), TNF-α (TNF) and TNF-R1 (p55/TNFRSF1A) (Table 2). CD40 is a TNF receptor family member that is widely recognized for its prominent role in immune regulation and homeostasis [57]. However, accumulating evidence suggests that the CD40 pathway can be exploited for cancer therapy as it can stimulate the host's anti-tumour immune response, followed by normalisation of the tumour microenvironment and arrest of the growth of CD40-positive tumours [57]. Further, CD40 also contains a cytoplasmic motif reminiscent of the death domain which is involved in the initiation of TNF-R1 and CD95-dependent apoptosis, stimulating cell death in cells of mesenchyme origin, tumour cells and certain transformed cell lines [58–60]. Moreover, membrane- anchored TNF-α and TNF-R1 (p55) may be activated by CD40 followed by stimulation of the death receptor-dependent pathway involving caspases-8 and -3 [61].

In another pathway, death-associated protein kinase 1 (DAPK1), a tumour suppressor, was highly expressed by 1 in HepG2 cells (by about 43-fold; Table 2). Over-expression of siDAPK1 significantly rescues the protein expression of transcription factor Rel/nuclear factor-kappaB (NF-κB)-targeted genes [62]. NF- B plays a crucial role in regulating gene transcription and is involved in the mechanism of cell proliferation [63,64]. DAPK1 also suppresses protein expression of anti-proliferation genes such as COX-2 and ICAM-1, and anti-apoptosis genes such as XIAP, which is involved in apoptosis inhibition triggered by multiple varied stimuli that activate both of the principle cell death pathways [65]. XIAP is an efficient apoptotic inhibitor because it is critically positioned to act at the point of convergence of both the extrinsic

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BFAR	**1.55**	**2.88**
BID	**13.37**	**2.16**
BIK	**7.52**	**8.22**
NAIP	**24.44**	**67.11**
BIRC2	*−1.35*	**1.85**
BIRC3	*−185.95*	*−30.94*
XIAP	*−68.65*	**4.90**
BIRC6	*−56.06*	*−24.28*
BIRC8	*−18417.65*	*−4842.45*
BNIP1	**75.12**	**179.58**
BNIP2	**227.73**	**595.72**
CASP3	**97.69**	**4.91**
CASP4	**8.64**	**9.31**
CASP5	**3.41**	**3.36**
CASP6	**38.35**	**109.78**
CASP7	**27.69**	**159.62**
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TP73	**18.79**	**8.62**
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TRAF3	*−3.14*	**73.46**
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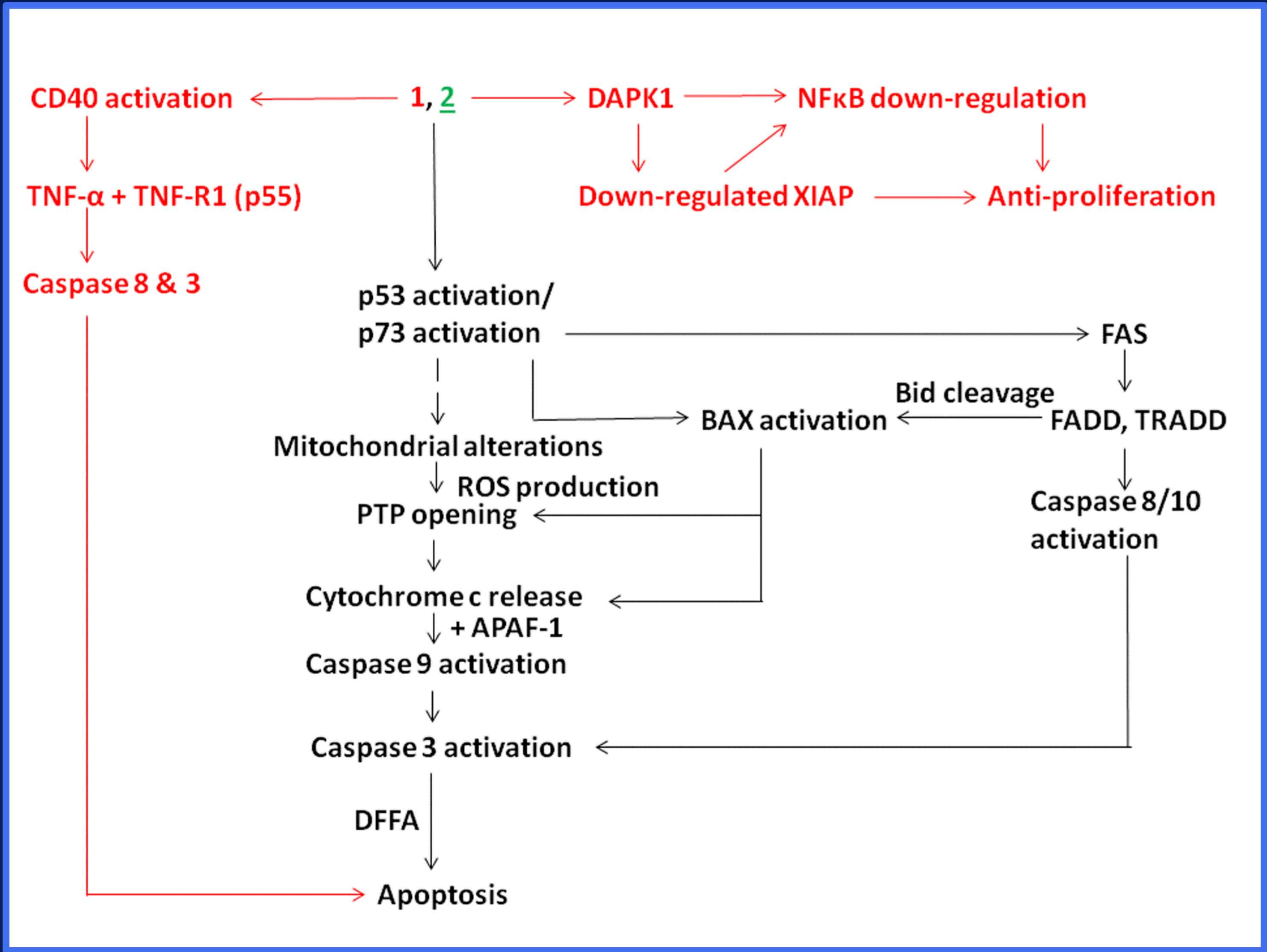
[a] Data represent mean of samples 1 and 2-induced fold-change in gene expression relative to control-treated cells (n = 3), $p < 0.05$.

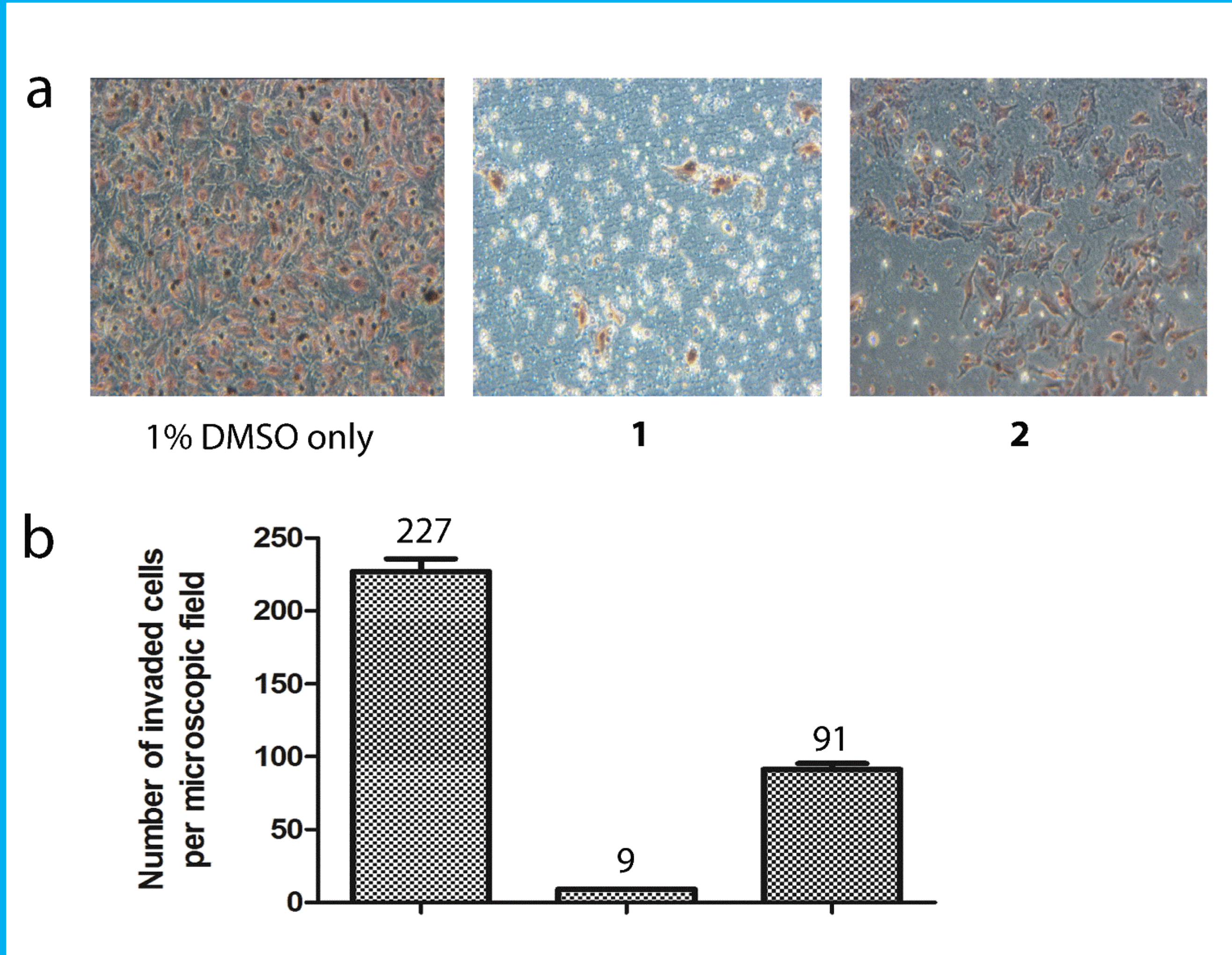
receptors of the tumour necrosis factor receptor superfamily, member 6 (FAS) and the tumour necrosis factor receptor (TNFRSF1A) gene expression. Caspases-8, -10, -9 and -3 were activated subsequently after FAS expression with an increase in their enzymatic activities (Tables 2–4). In general, FAS and TNFR1 recruit Fas-associated protein with death domain (FADD) and procaspase-8 and -10 to the receptor. FADD controls the recruiting of procaspase-8 and -10 leading to its auto-cleavage and activation, and subsequently activates effector caspases in initiating cell death [52,53]. Alternatively, caspase-8 and -10 could cleave the BCL-2 family member Bid, a molecular linker bridging death receptor

receptor-dependent pathway by induction of a series of pro-apoptotic genes, such as CD40 (CD40 gene), CD40L (CD40LG), TNF-α (TNF) and TNF-R1 (p55/TNFRSF1A) (Table 2). CD40 is a TNF receptor family member that is widely recognized for its prominent role in immune regulation and homeostasis [57]. However, accumulating evidence suggests that the CD40 pathway can be exploited for cancer therapy as it can stimulate the host's anti-tumour immune response, followed by normalisation of the tumour microenvironment and arrest of the growth of CD40-positive tumours [57]. Further, CD40 also contains a cytoplasmic motif reminiscent of the death domain which is involved in the initiation of TNF-R1 and CD95-dependent apoptosis, stimulating cell death in cells of mesenchyme origin, tumour cells and certain transformed cell lines [58–60]. Moreover, membrane- anchored TNF-α and TNF-R1 (p55) may be activated by CD40 followed by stimulation of the death receptor-dependent pathway involving caspases-8 and -3 [61].

In another pathway, death-associated protein kinase 1 (DAPK1), a tumour suppressor, was highly expressed by 1 in HepG2 cells (by about 43-fold; Table 2). Over-expression of siDAPK1 significantly rescues the protein expression of transcription factor Rel/nuclear factor-kappaB (NF-κB)-targeted genes [62]. NF- B plays a crucial role in regulating gene transcription and is involved in the mechanism of cell proliferation [63,64]. DAPK1 also suppresses protein expression of anti-proliferation genes such as COX-2 and ICAM-1, and anti-apoptosis genes such as XIAP, which is involved in apoptosis inhibition triggered by multiple varied stimuli that activate both of the principle cell death pathways [65]. XIAP is an efficient apoptotic inhibitor because it is critically positioned to act at the point of convergence of both the extrinsic



CD40 activation
1, 2
DAPK1
NFκB down-regulation
TNF-α + TNF-R1 (p55)
Down-regulated XIAP
Anti-proliferation
Caspase 8 & 3
p53 activation/
p73 activation
FAS
Bid cleavage
BAX activation
FADD, TRADD
Mitochondrial alterations
ROS production
PTP opening
Caspase 8/10
activation
Cytochrome c release
+ APAF-1
Caspase 9 activation
Caspase 3 activation
DFFA
Apoptosis



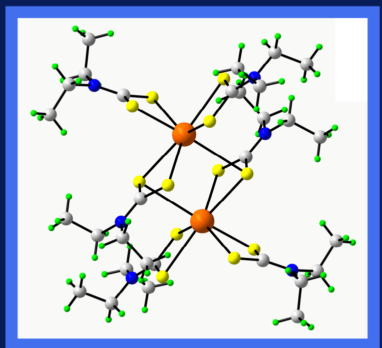
Cell cycle analysis, ROS, cytochrome C, cell invasion…

Killing Helicobacter pylori

MIC_{90} (μg/ml)

Average of 16 strains

Et	$N(CH_2)_4$	*n*-Bu	BSS
4	8	256	16



with A/Prof Ho Bow & Sook Yin Lui

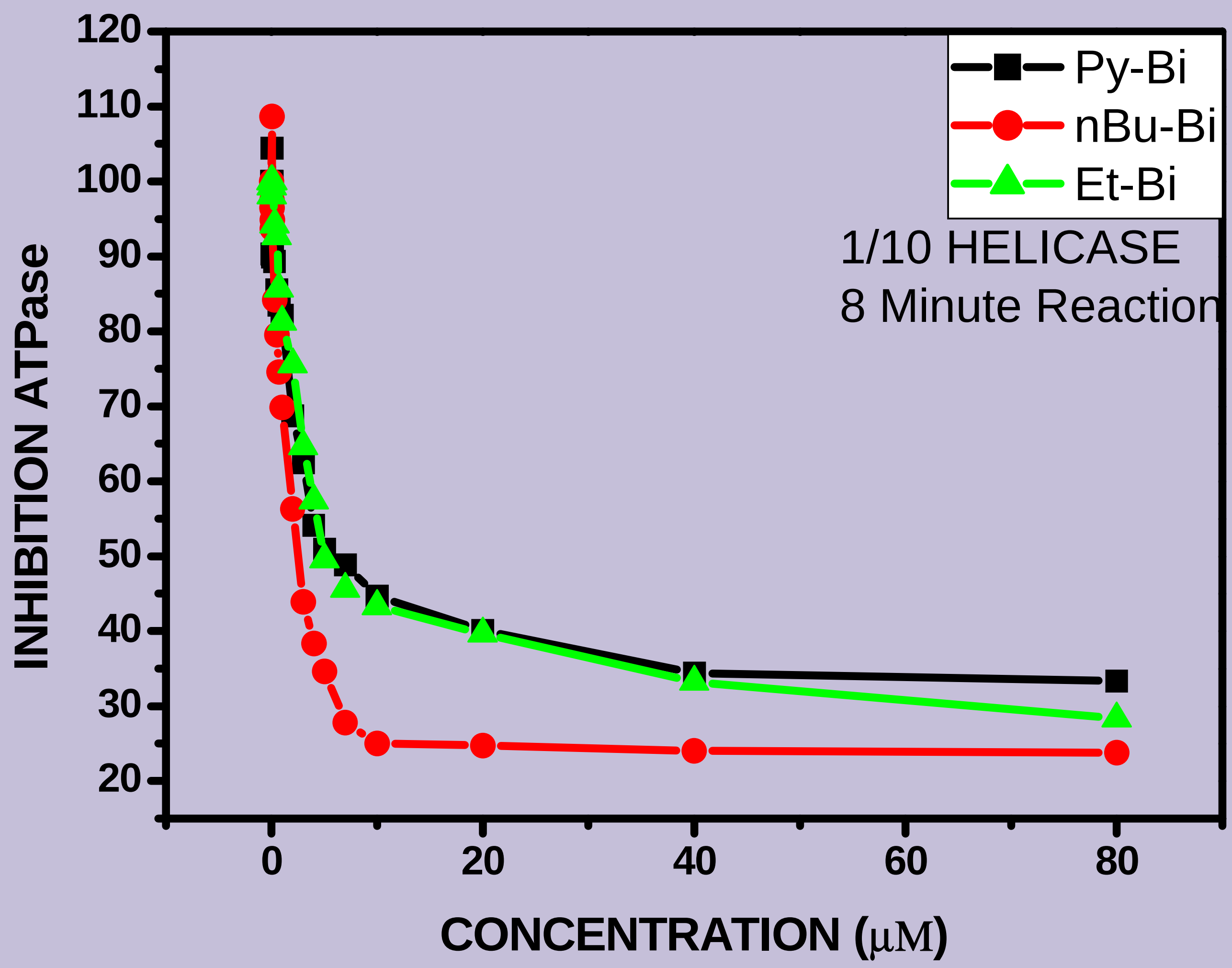
Avian Flu

Severe Acute Respiratory Syndrome

RNA virus

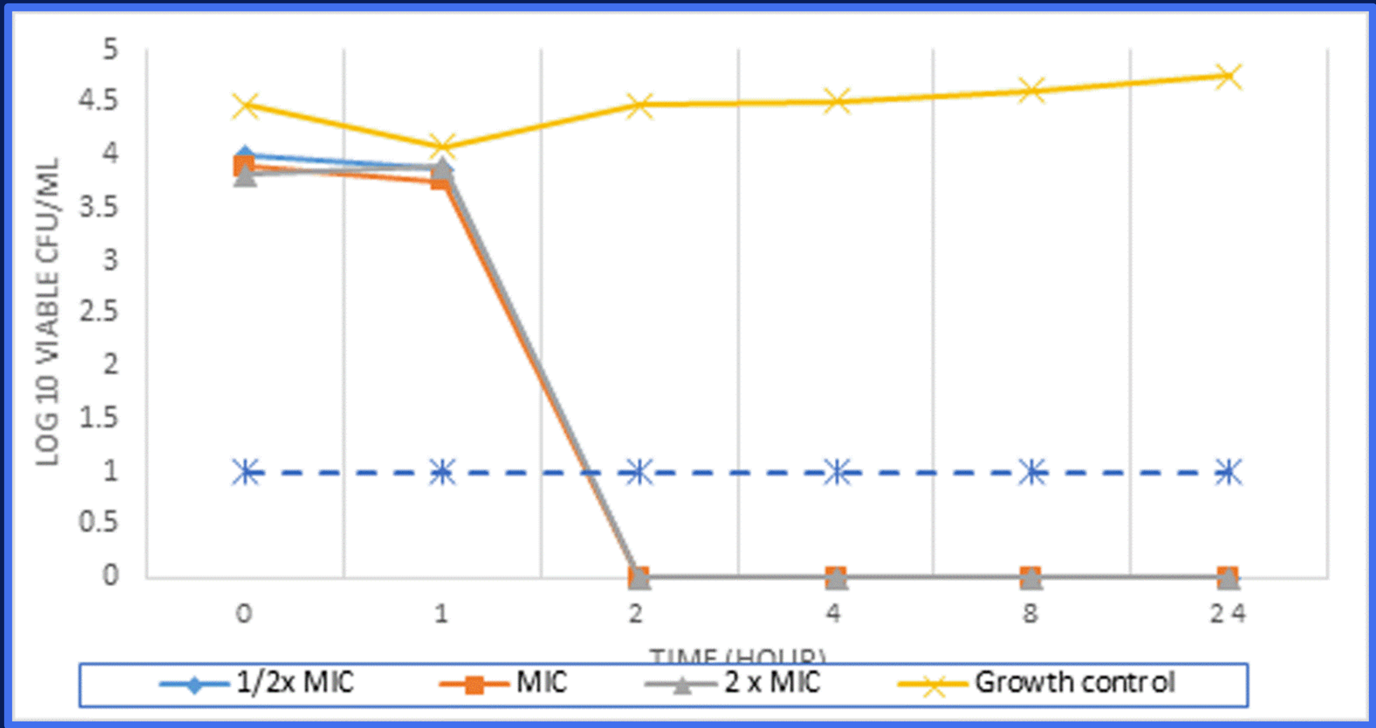
http://www.smh.com.au

http://www.germbusters.com.sg



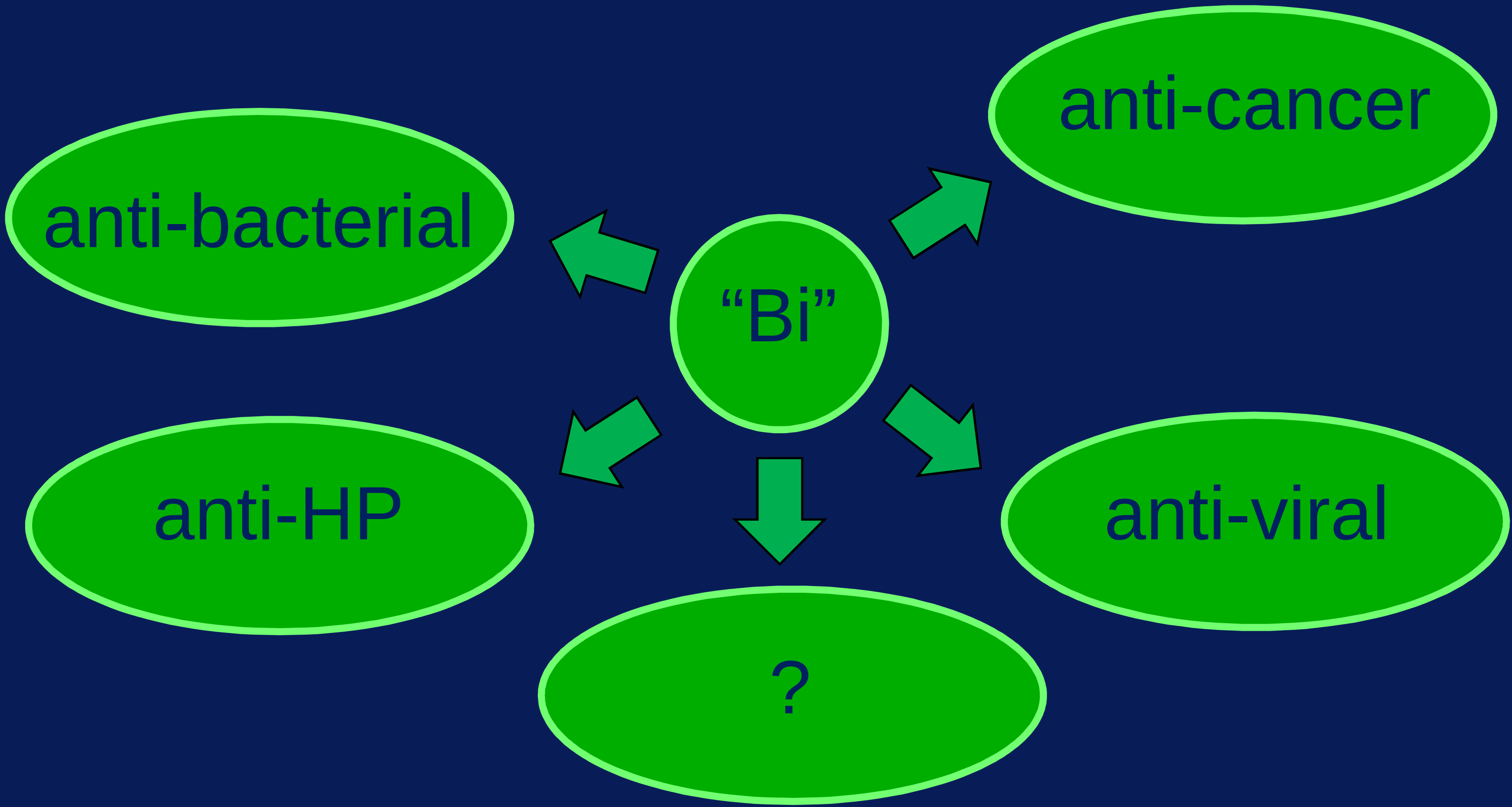
Sun Hongzhe *et al*.

Anti-bacterial activity



Bactericidal against *Streptococcus pneumoniae*

Bismuth-Based Drugs



Exciting potential for medicine with important advances waiting to be made!

Why molecules pack as they do?

1,1-dithiolates

Biological bismuth

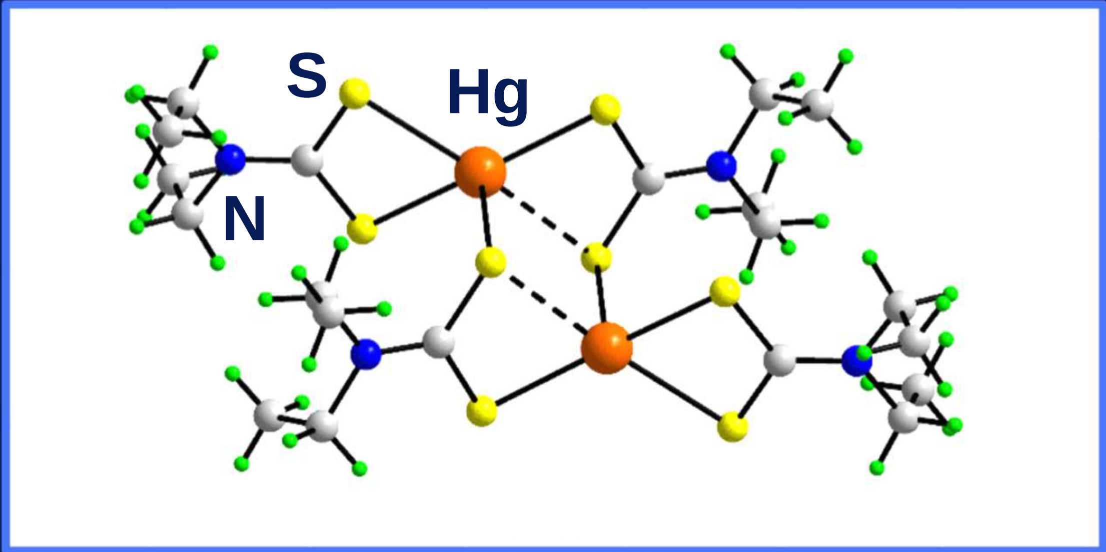
Secondary bonding

Extended architectures mediated by bipyridyl bridges

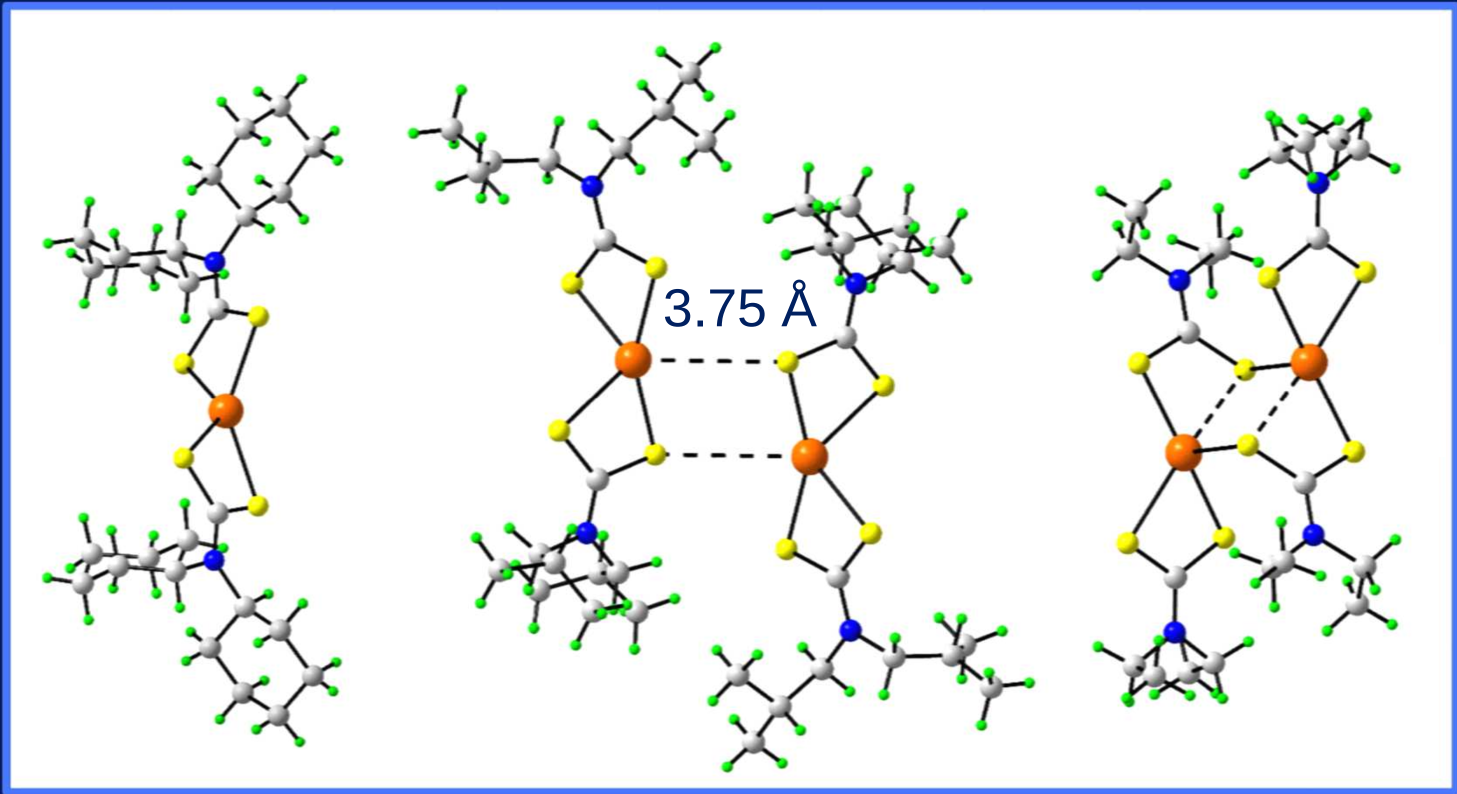
Perplexing results

Intermolecular interactions involving chelate rings

Structure of $Hg(S_2CNEt_2)_2$



Steric effects and secondary bonding



"$Hg(S_2CNR_2)_2$"

Sum of the van der Waals radii for Hg and S = 3.35 Å

← increasing size of R

Conclusion #1

Systematic analyses enables new design elements for crystal engineering

Crystal engineering?

Synthetic chemists: make molecules (covalent bonding)

CE's: design crystals (parts of crystals) by controlling intermolecular interactions

Conclusion #1

Systematic analyses enables new design elements for crystal engineering

Crystal engineering?

Synthetic chemists: make molecules (covalent bonding)

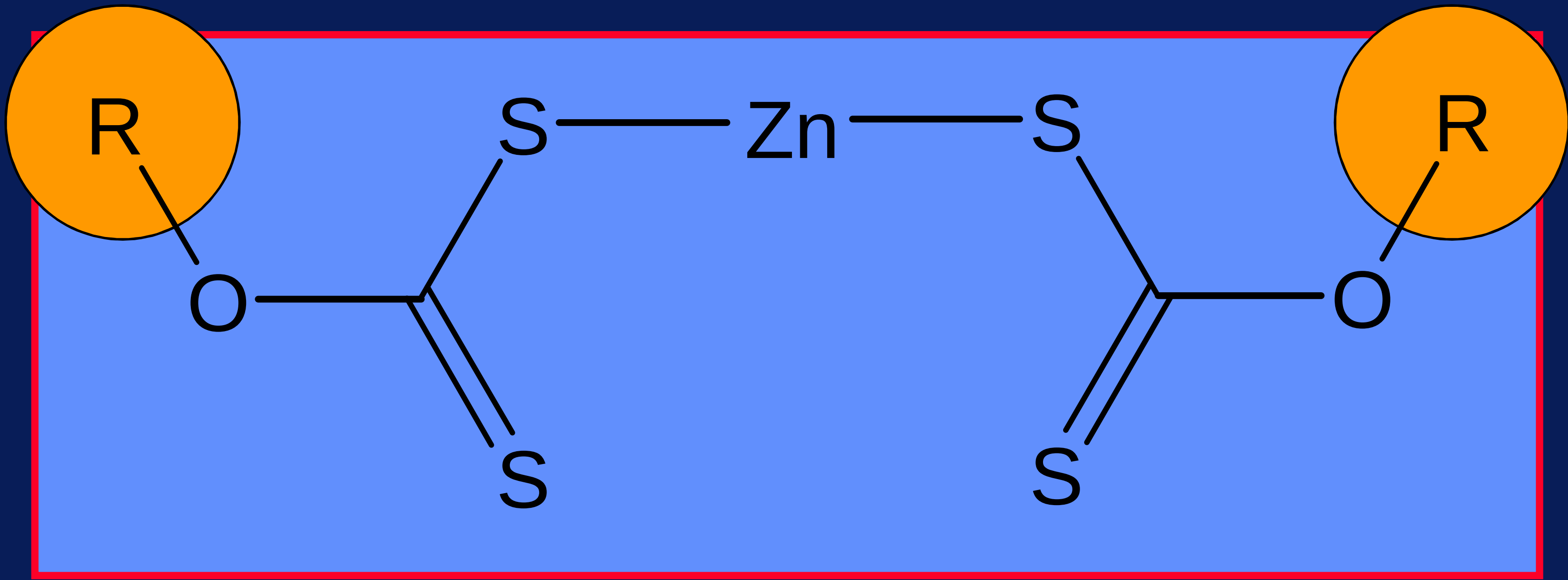
CE's: design crystals (parts of crystals) by controlling intermolecular interactions, e.g. hydrogen-bonding, halogen-bonding, secondary bonding, π-π, C-H…O, C-H…π, "emerging" interactions, etc.

Zinc thiolates: tuning supramolecular aggregation

Molecular paving with zinc xanthates

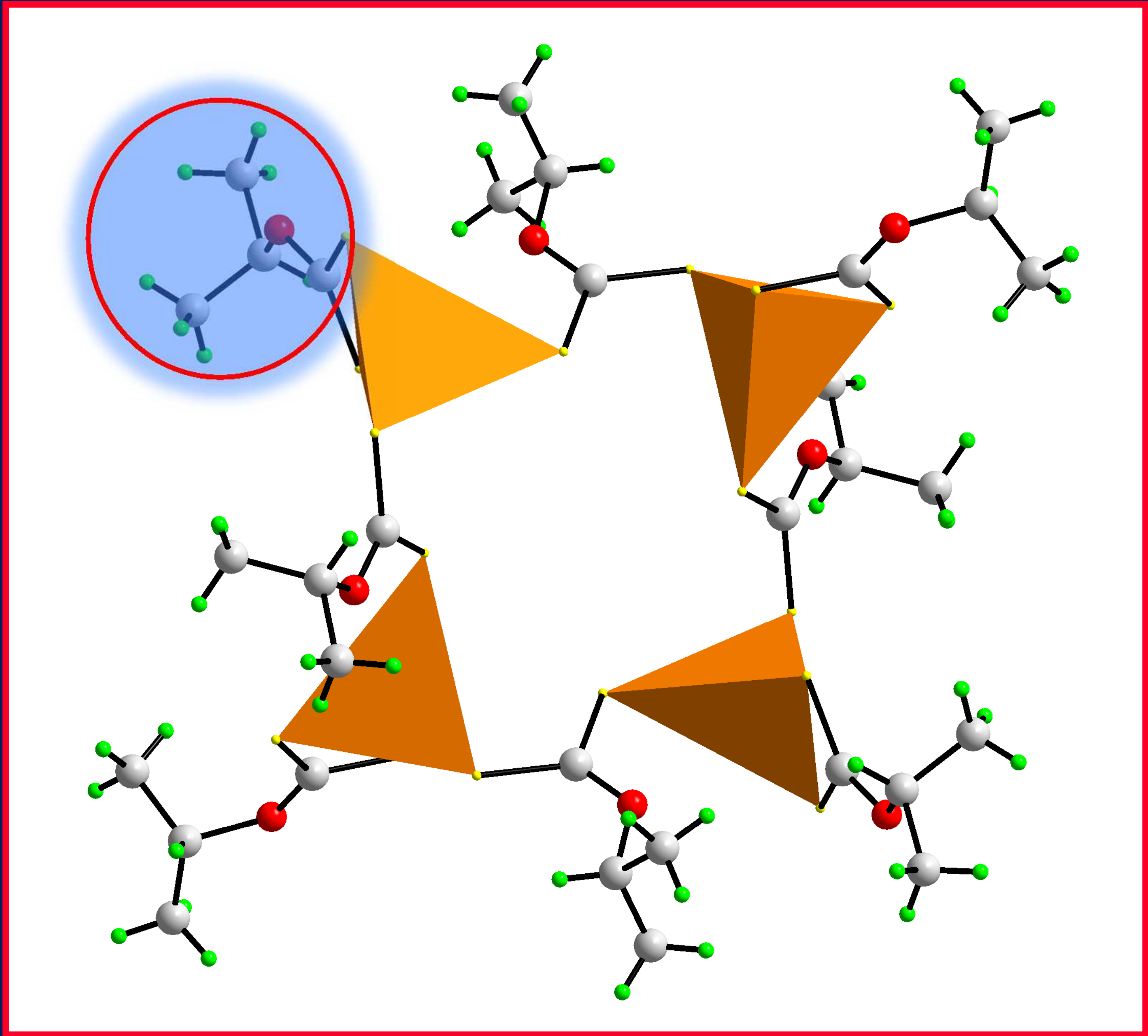
Tailoring luminescence

Structural diversity in $Zn(S_2COR)_2$



Examine structures for which R = Et, nPr & iPr

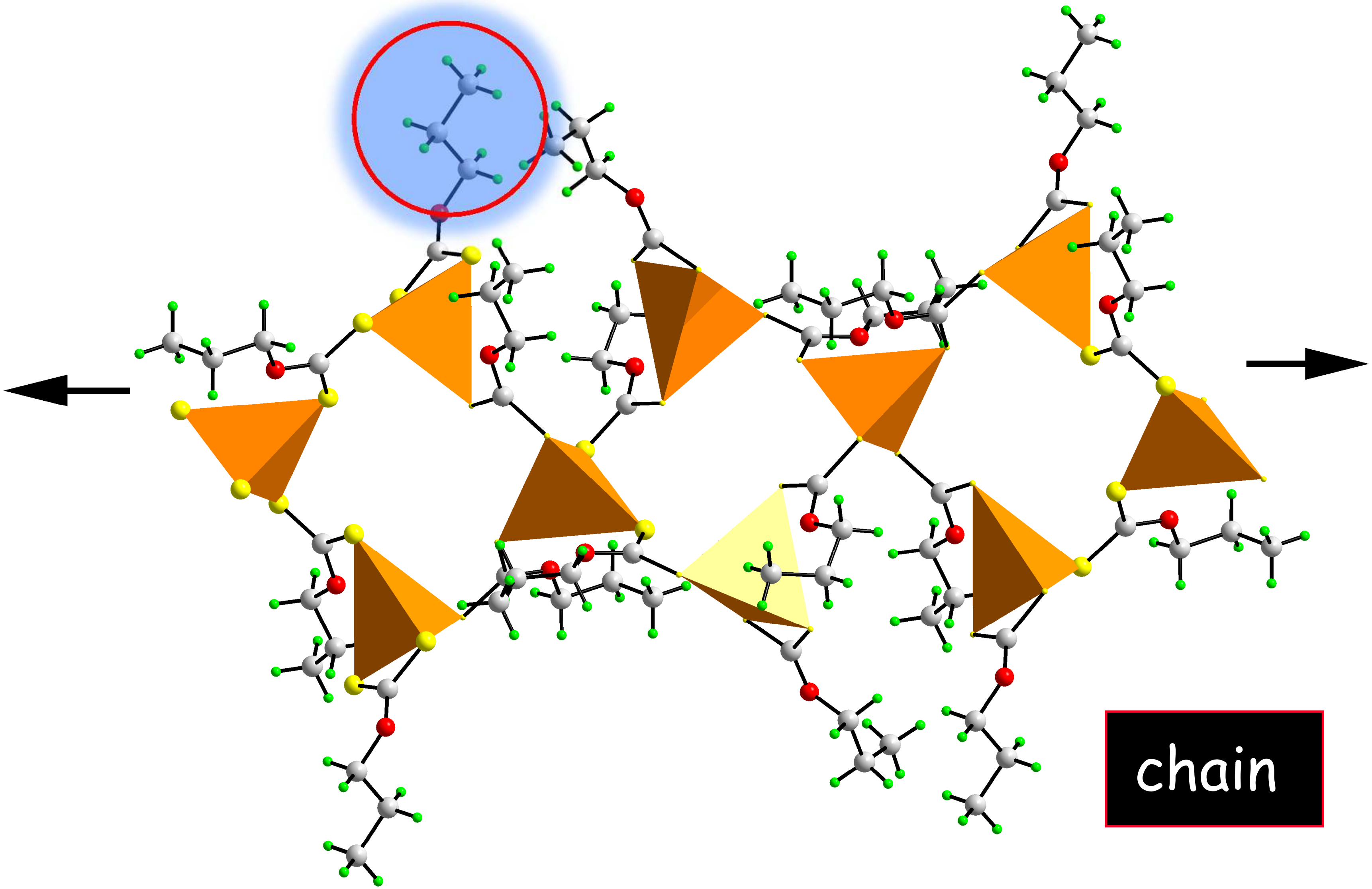
CrystEngComm , 2002, *4*, 596



R = iPr

isolated tetramer

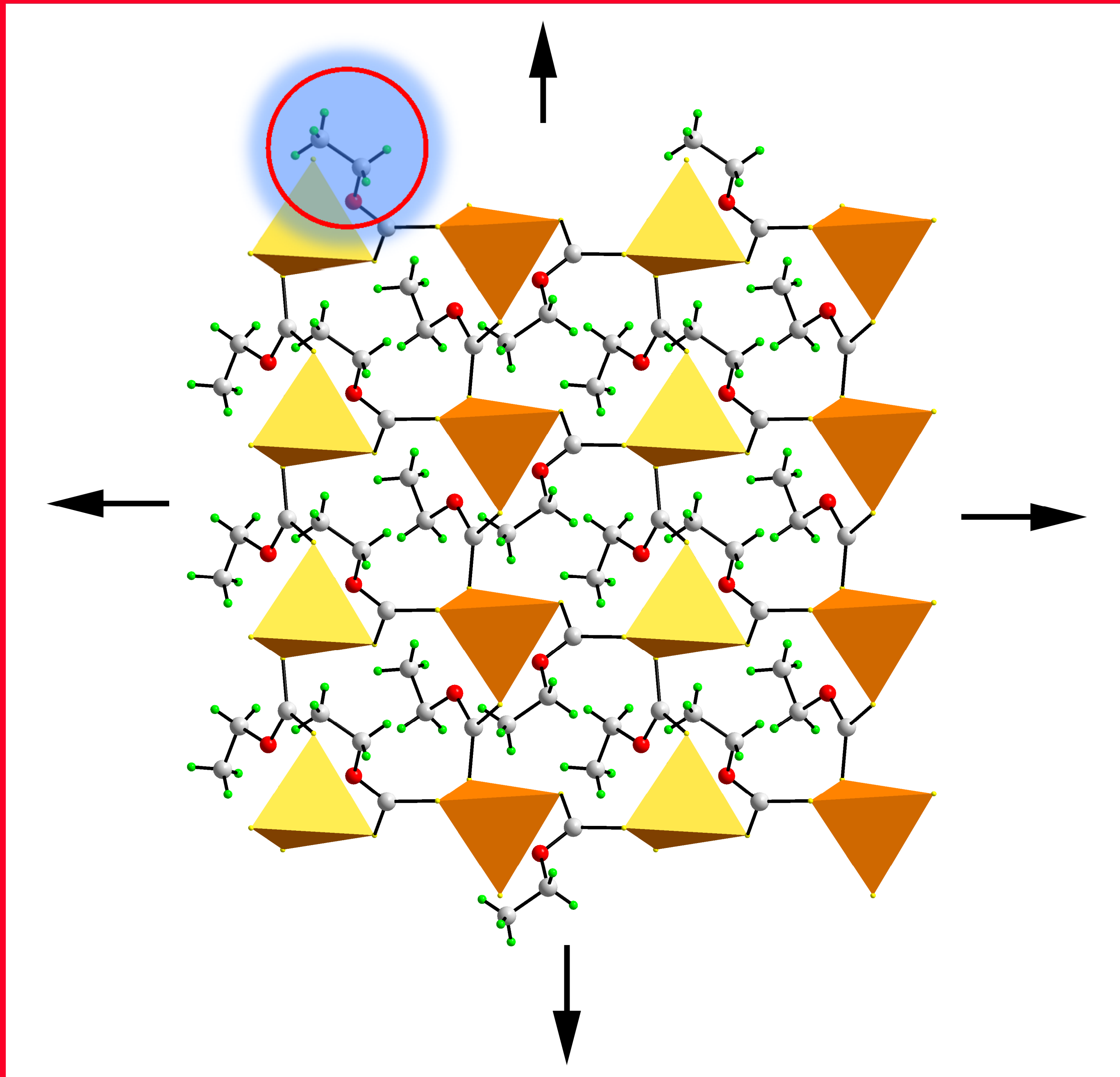
R = nPr



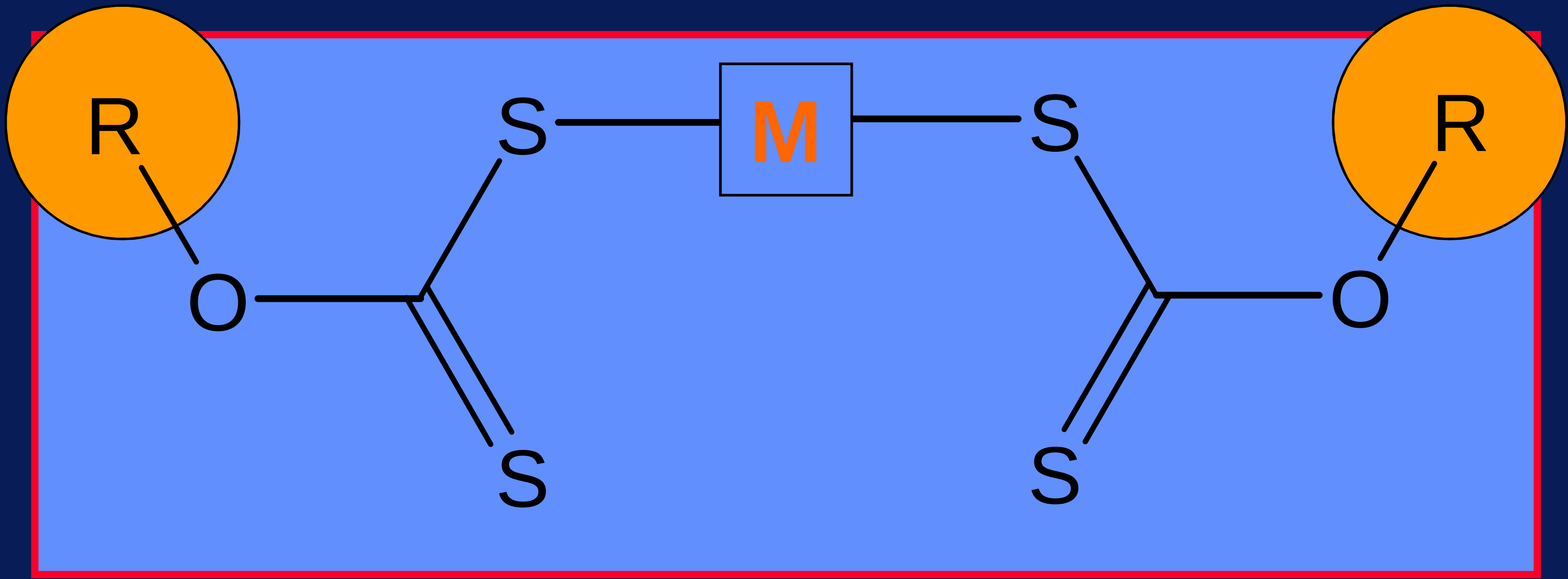
chain

R = Et

layer

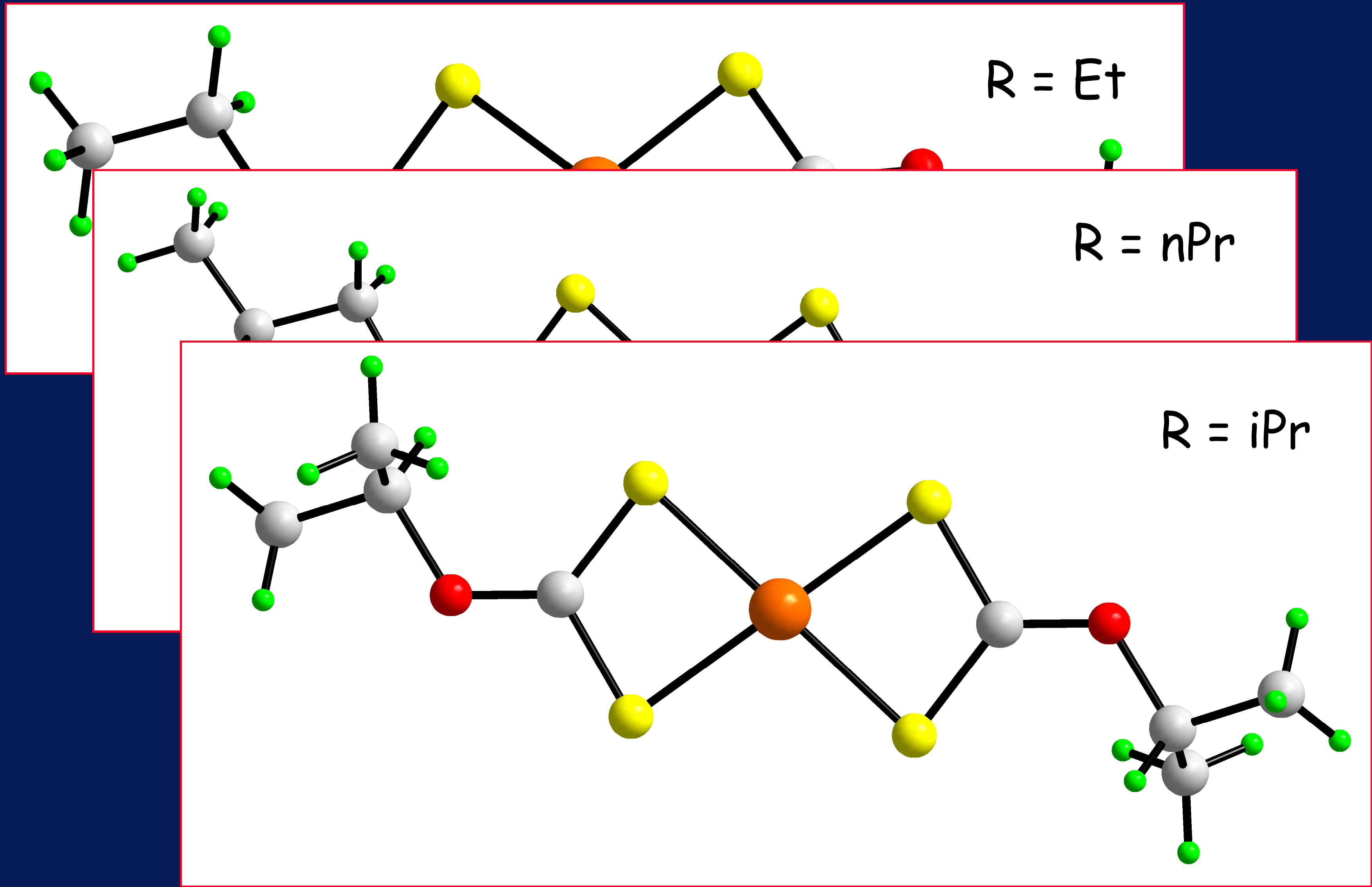


Structural diversity in $M(S_2COR)_2$

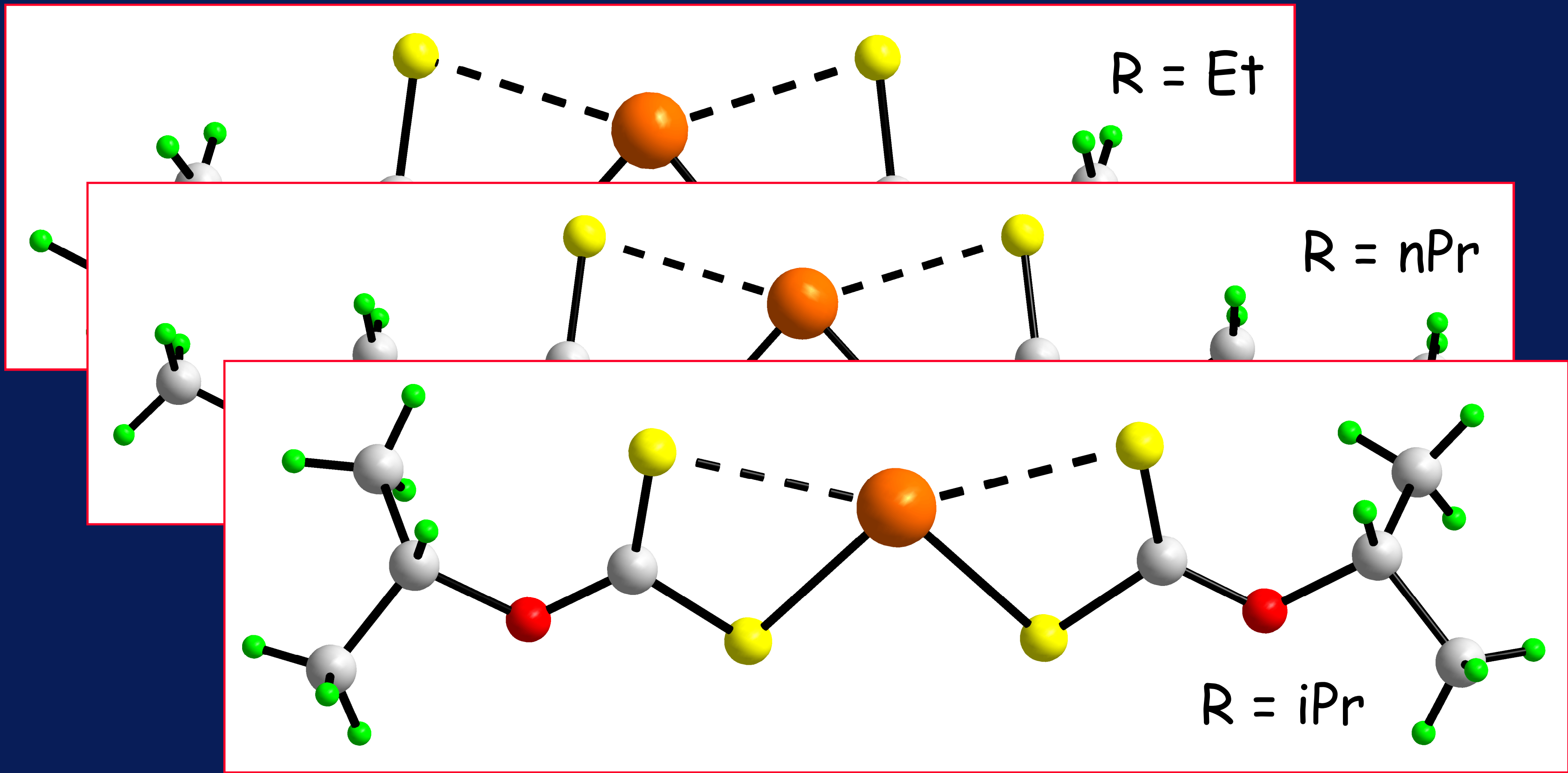


Examine structures for which R = Et, nPr & iPr

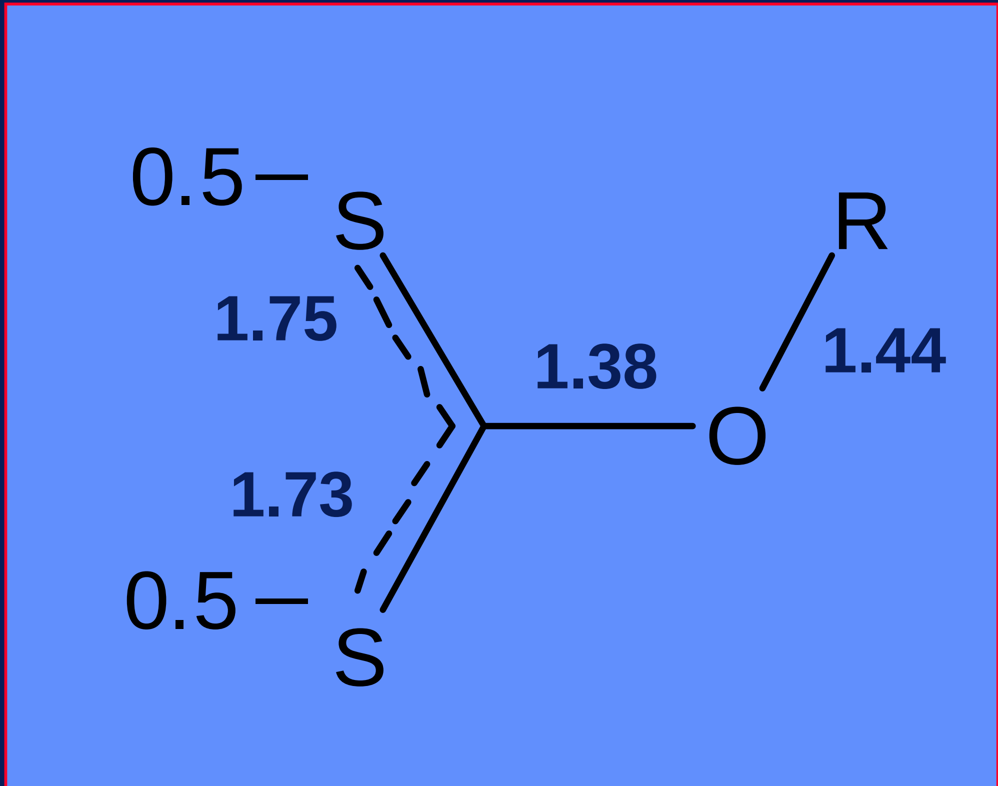
$Ni(S_2COR)_2$ for R = Et, nPr & iPr



$Te(S_2COR)_2$ for R = Et, nPr & iPr

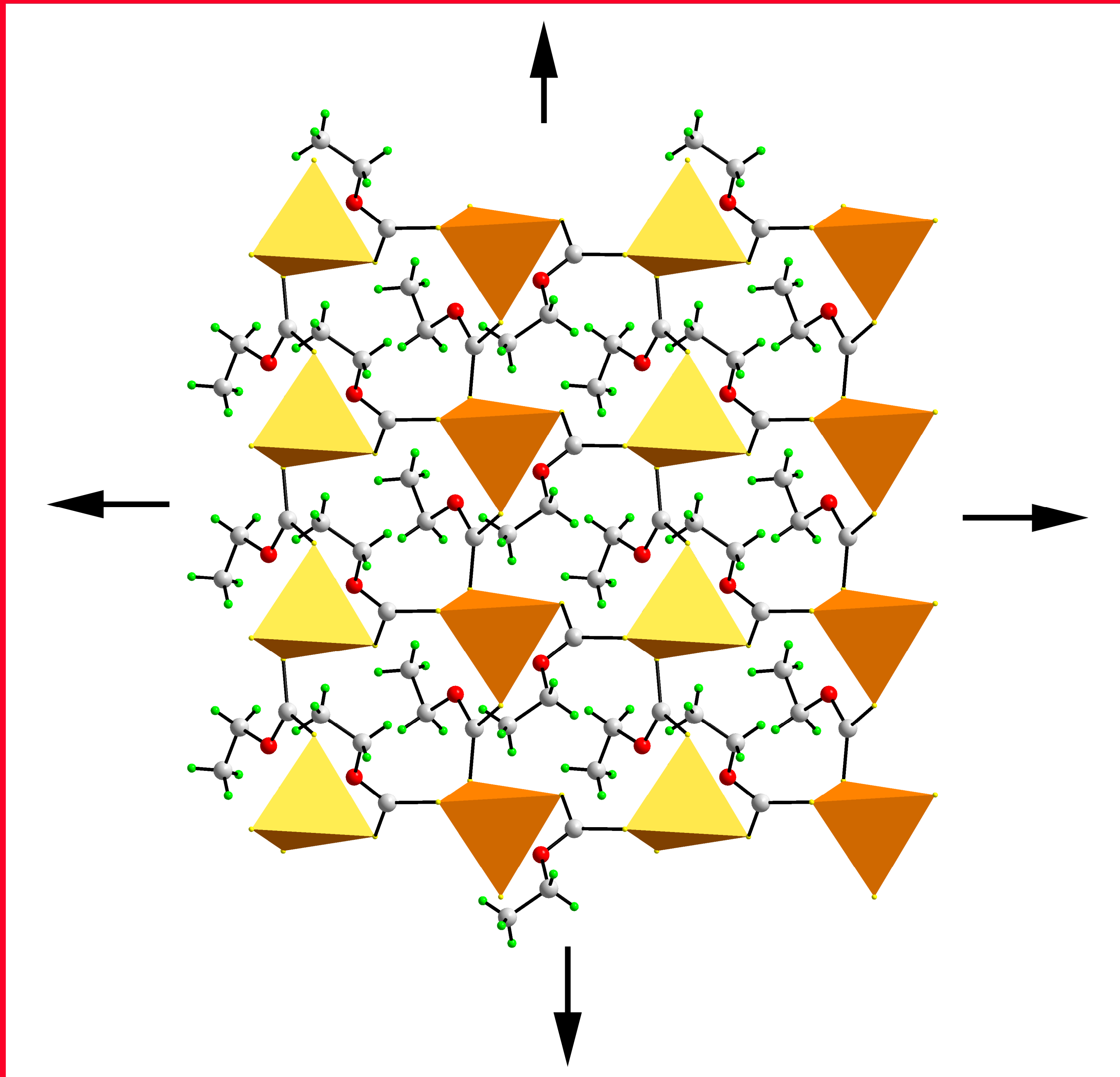


Ab initio molecular orbital calculations on $^{-}S_2COR$

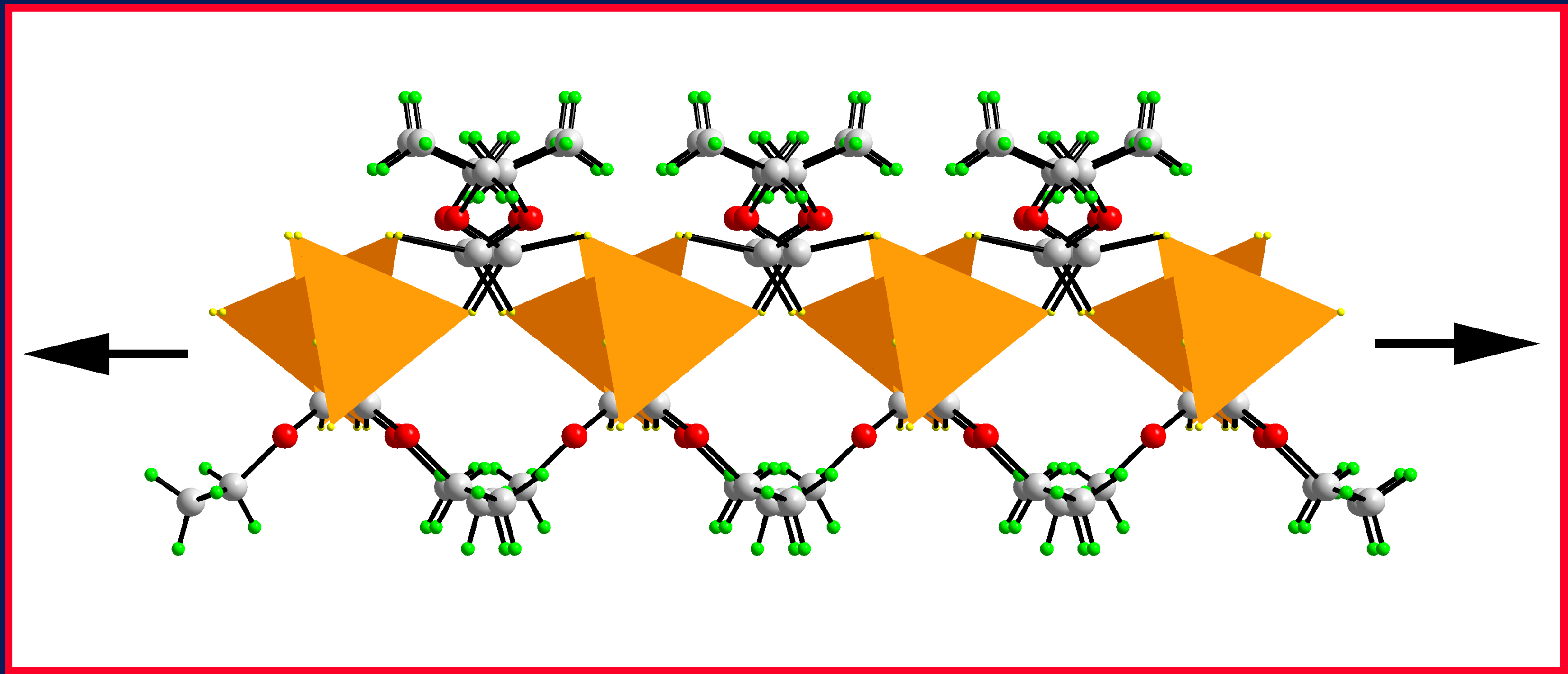


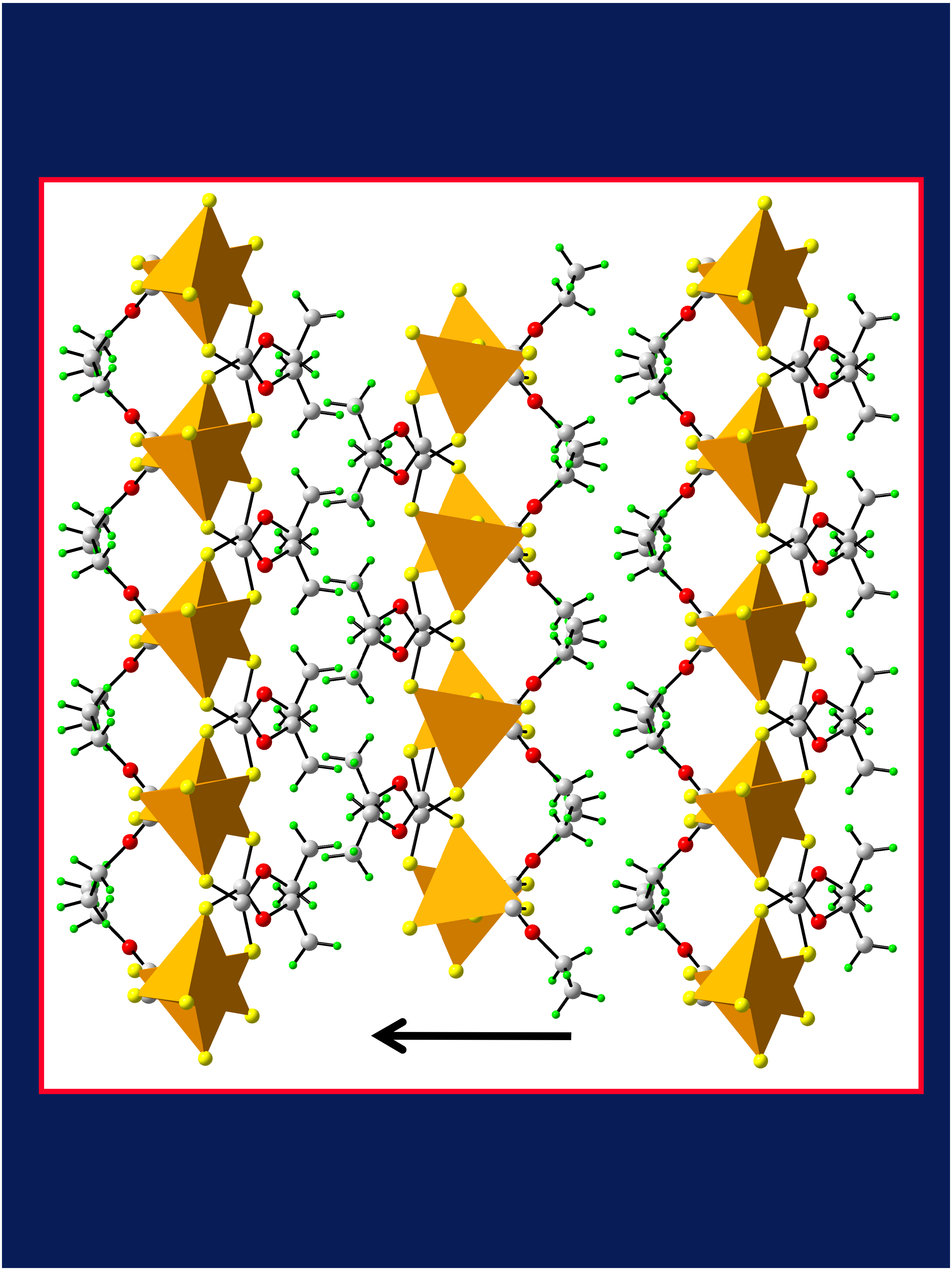
R = Et

layer

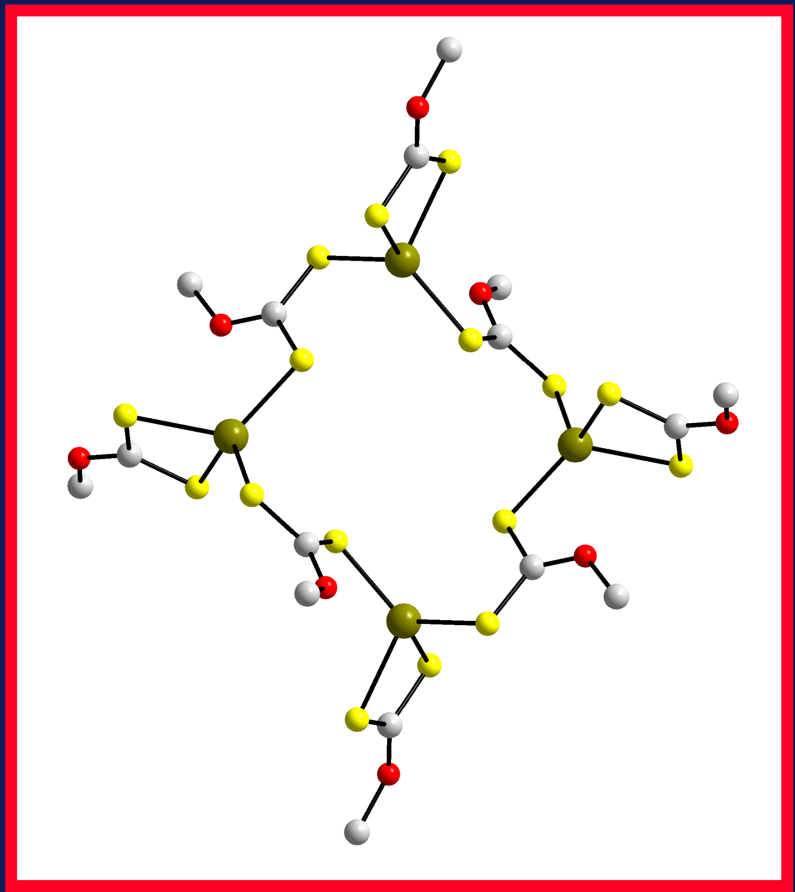


R = Et

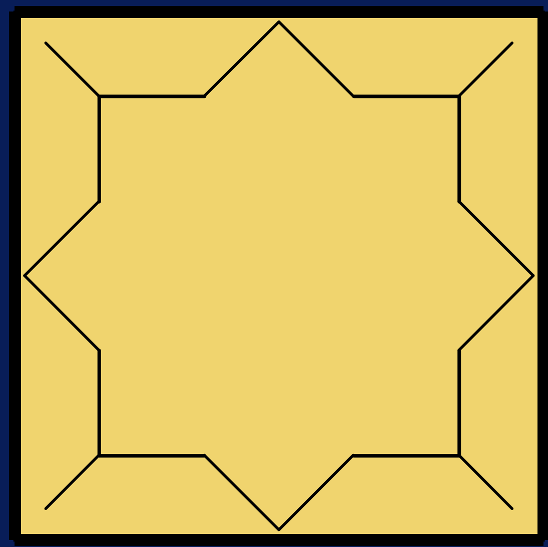




Molecular paving with $Zn(S_2COR)_2$

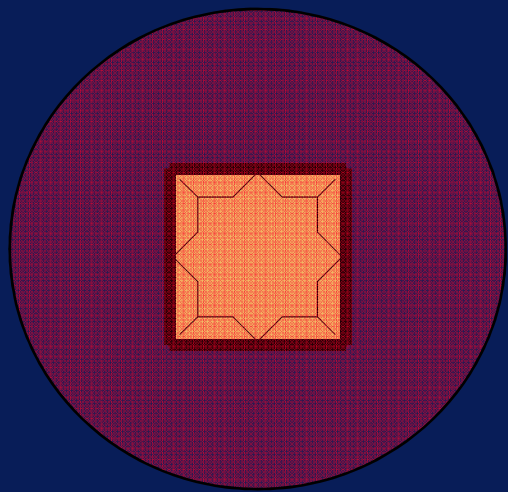


$\equiv$



'Molecular
Paving stone'

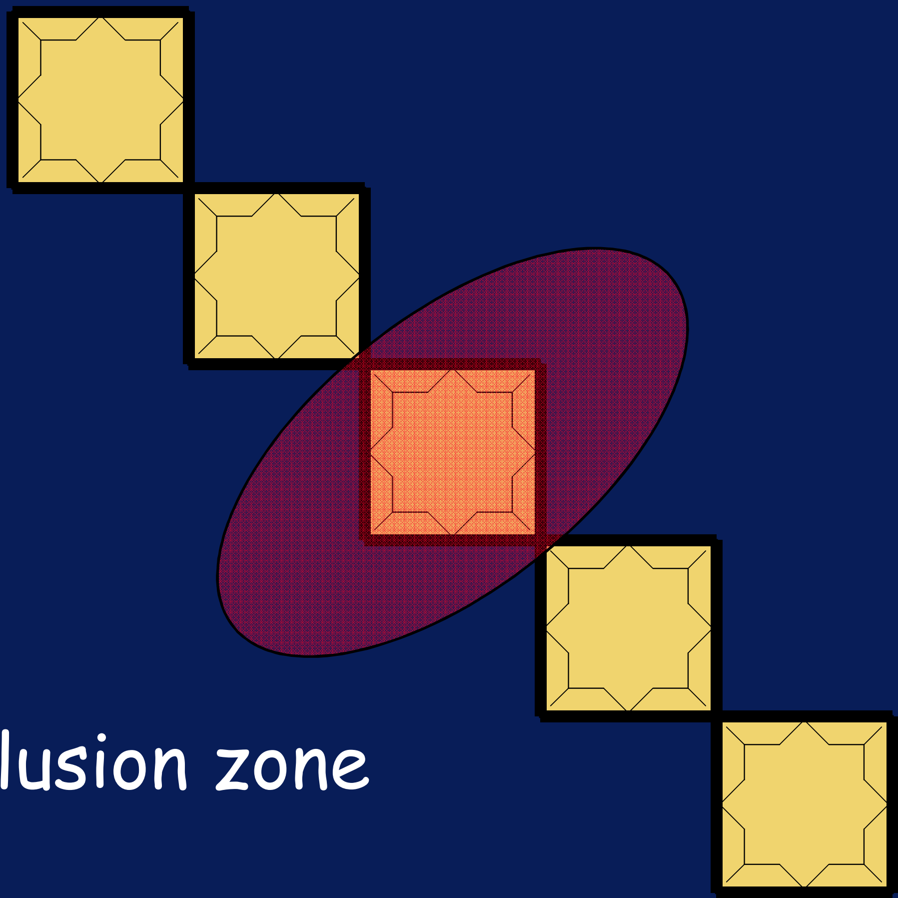
Molecular paving with $Zn(S_2COR)_2$



3-D exclusion zone

R =
i-Pr

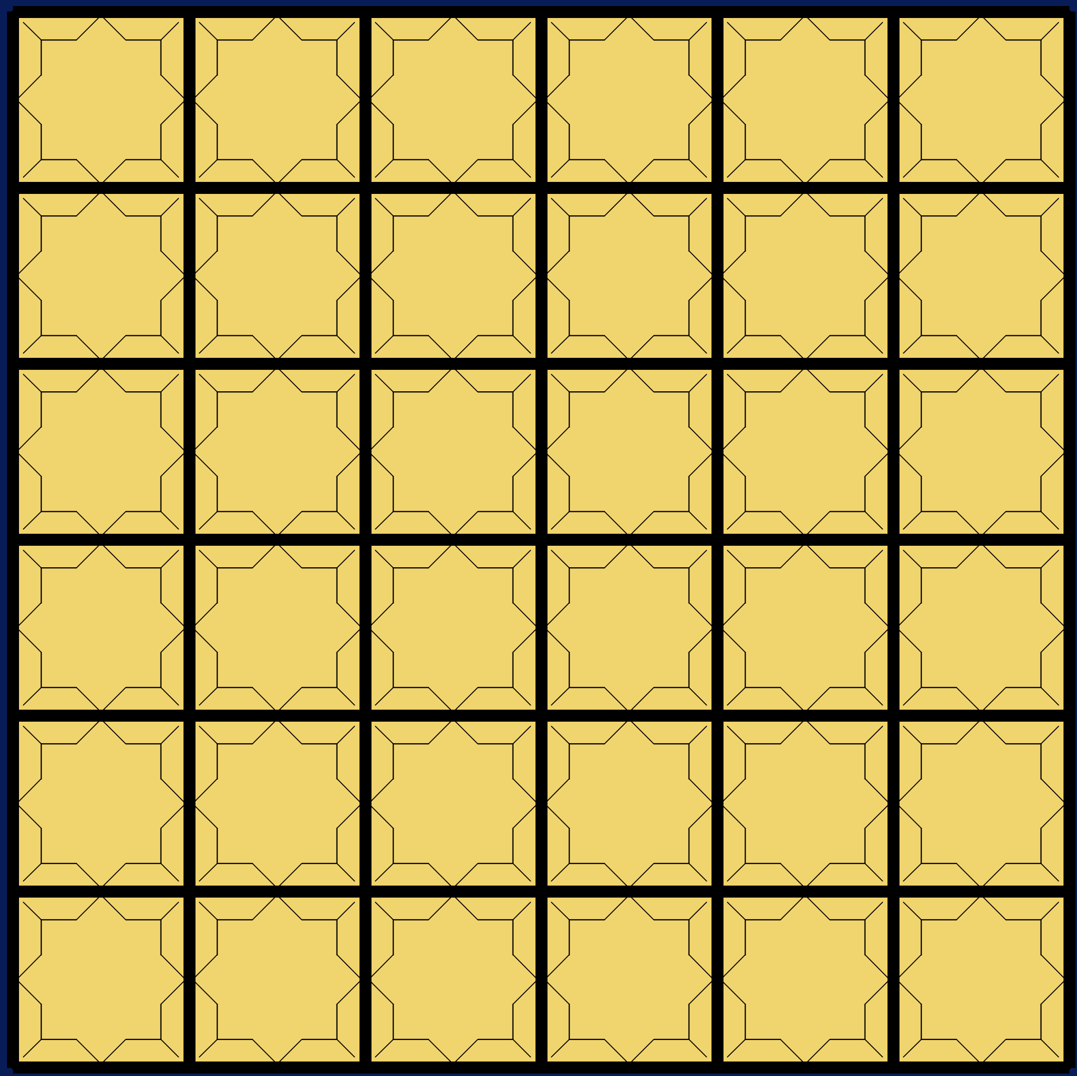
Molecular paving with $Zn(S_2COR)_2$



2-D exclusion zone

R = *n*-Pr

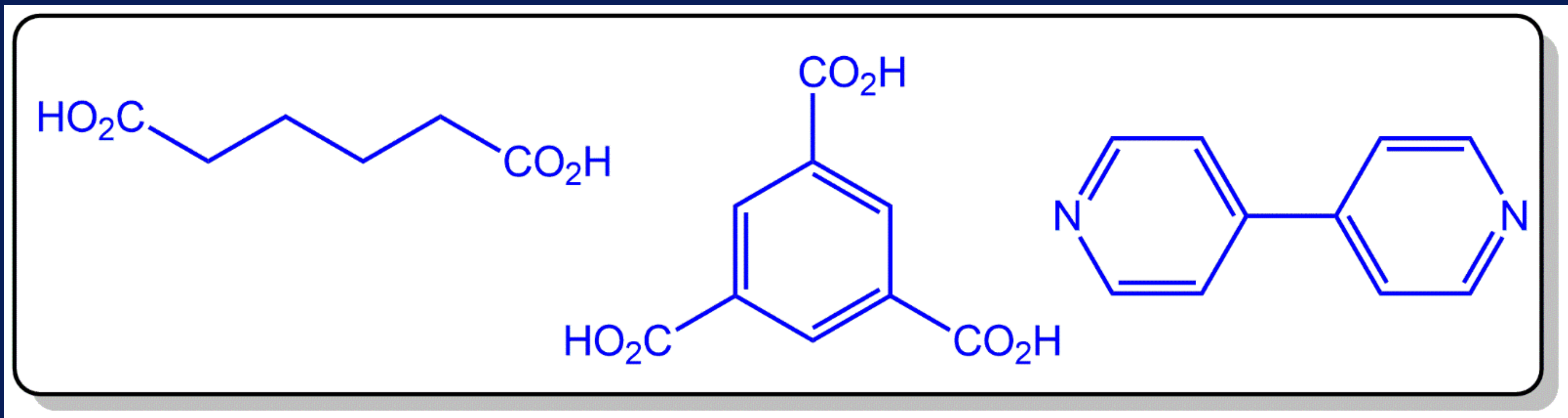
Molecular paving with $Zn(S_2COR)_2$



R = Et

Metal-organic frameworks & coordination polymers

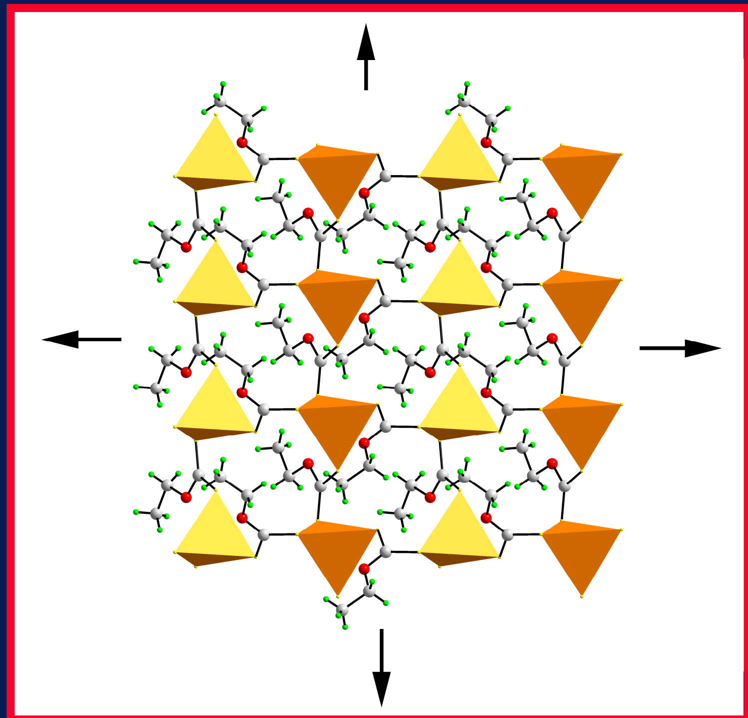
Applications: gas storage/sensing; catalysis;
luminescence; energy storage;
crystal sponge…



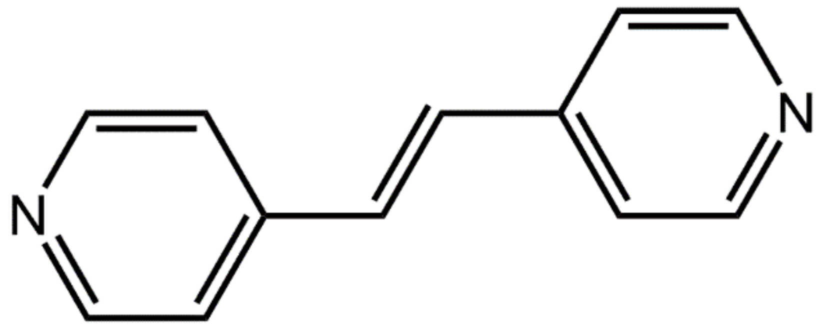
Coordination polymers of zinc-triad elements

Solid-state polymers cf. solution

$A(S_2COR)_2$ + bridging ligands

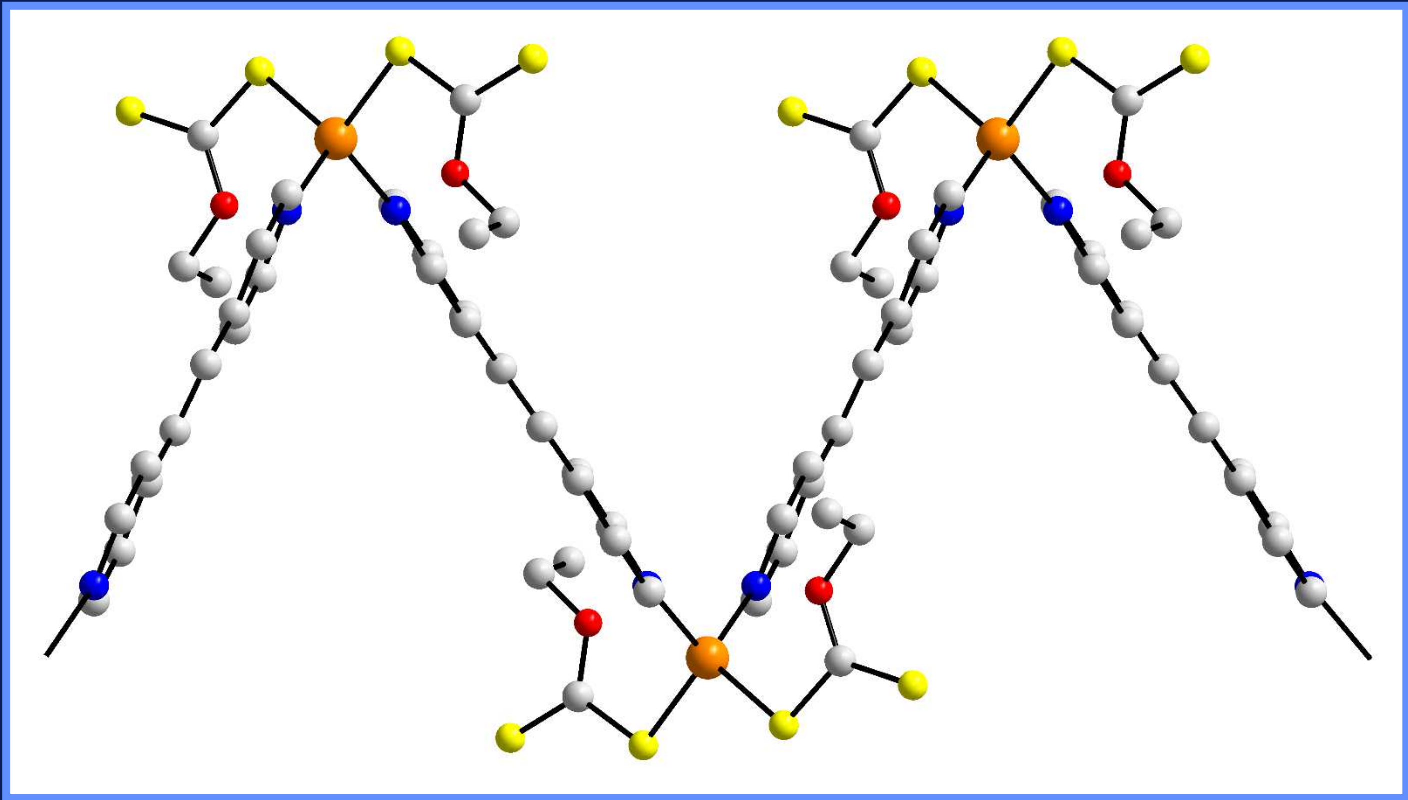


Zinc(1,1-dithiolate)$_2$ + bpe



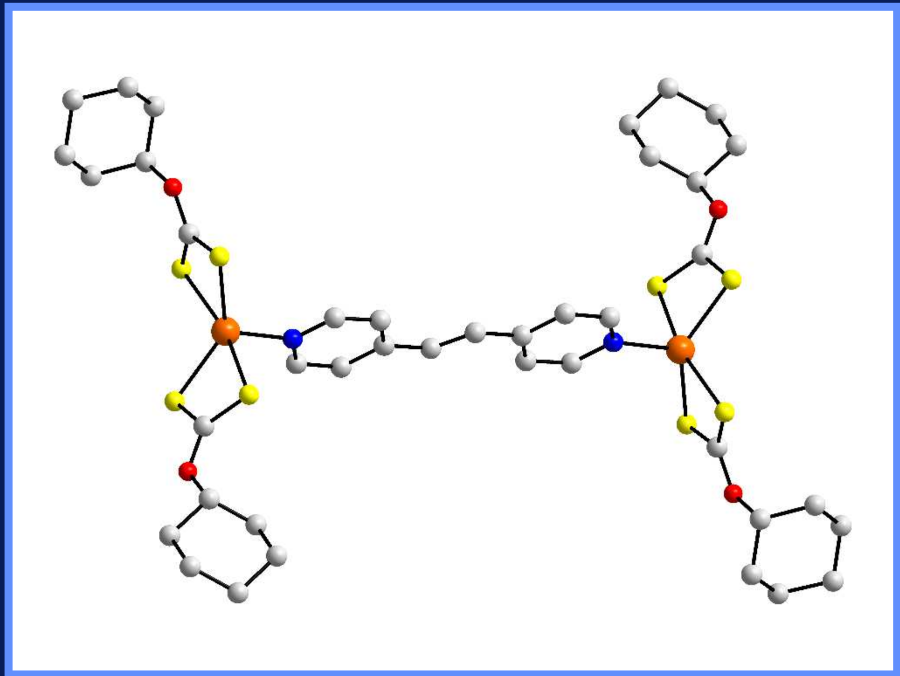
trans-1,2-bis(4-pyridyl)ethylene (bpe)

Zinc(xanthate)$_2$ + bpe



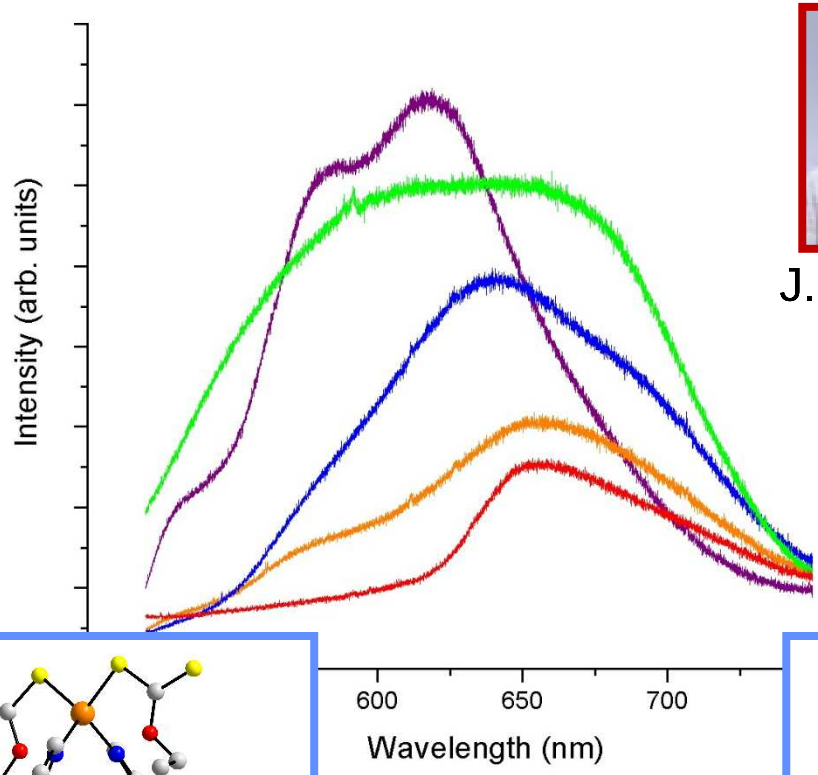
R = Et

Zinc(xanthate)$_2$ + bpe

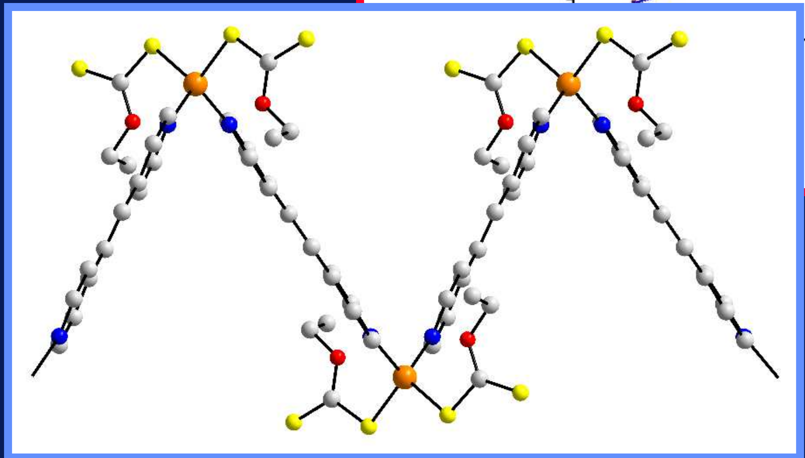


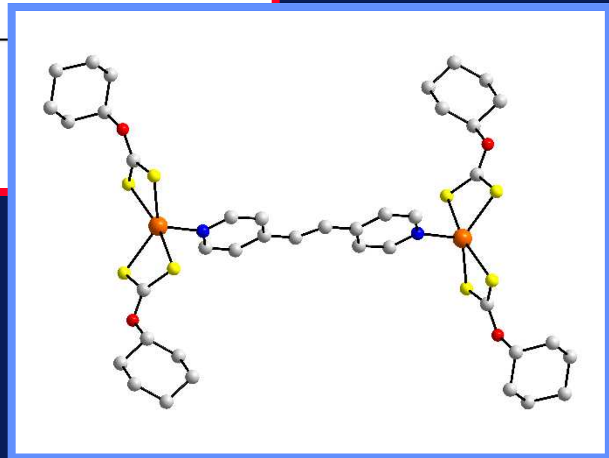
R = Cy

Implications for solid-state luminescence



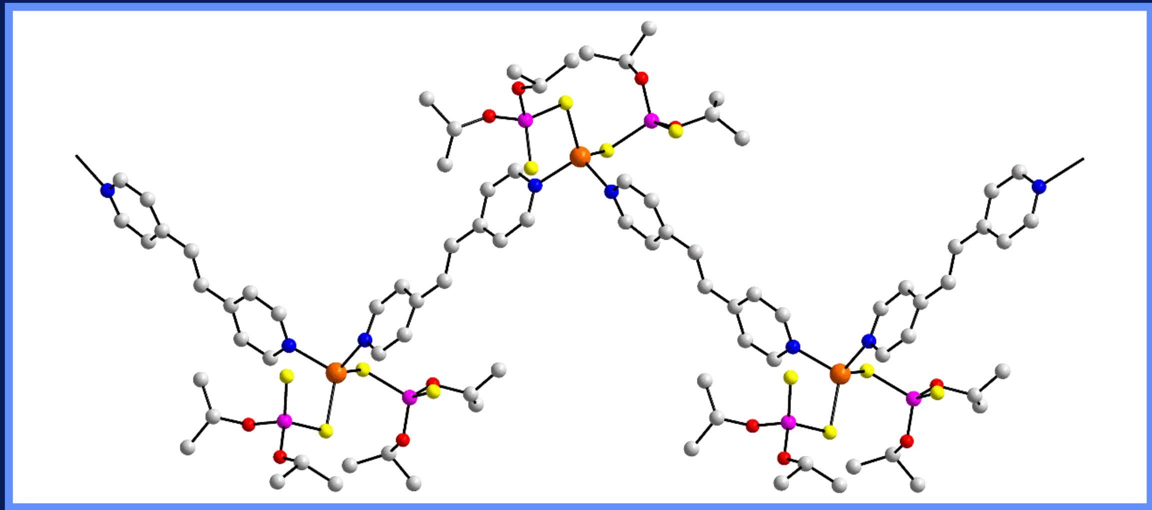
J.-G. Kang

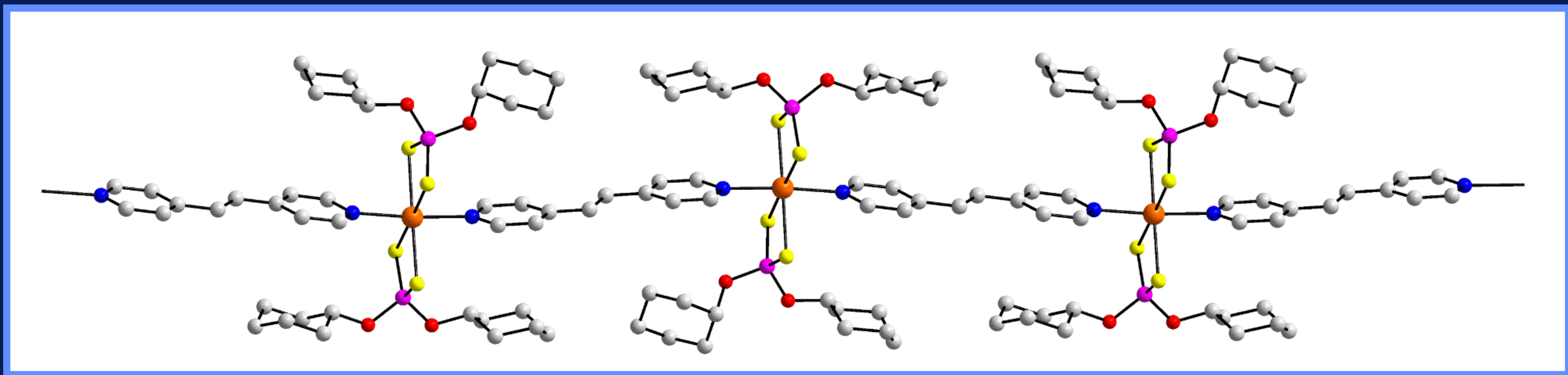




Zinc(dithiophosphate)$_2$ + bpe

R = iPr

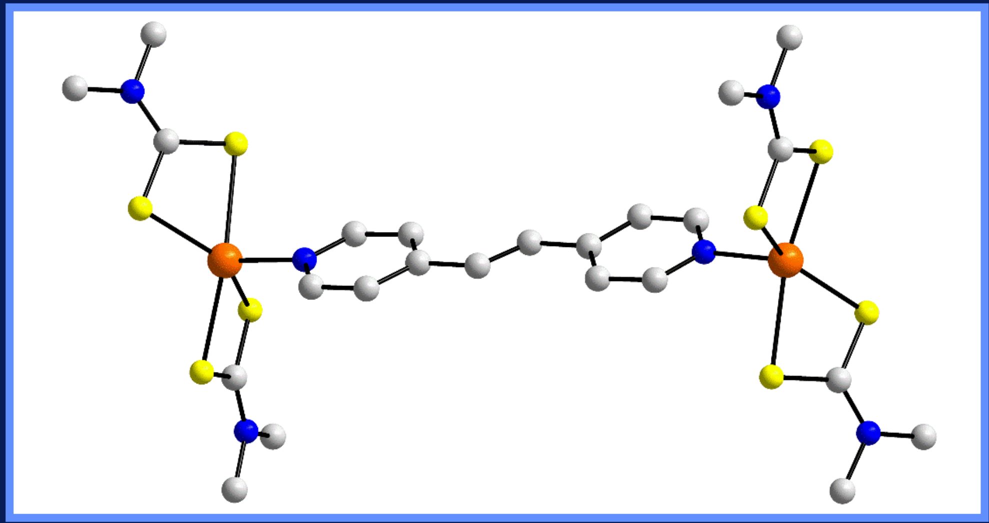




R = Cy

Zinc(dithiocarbamate)$_2$ + bpe

R = Me

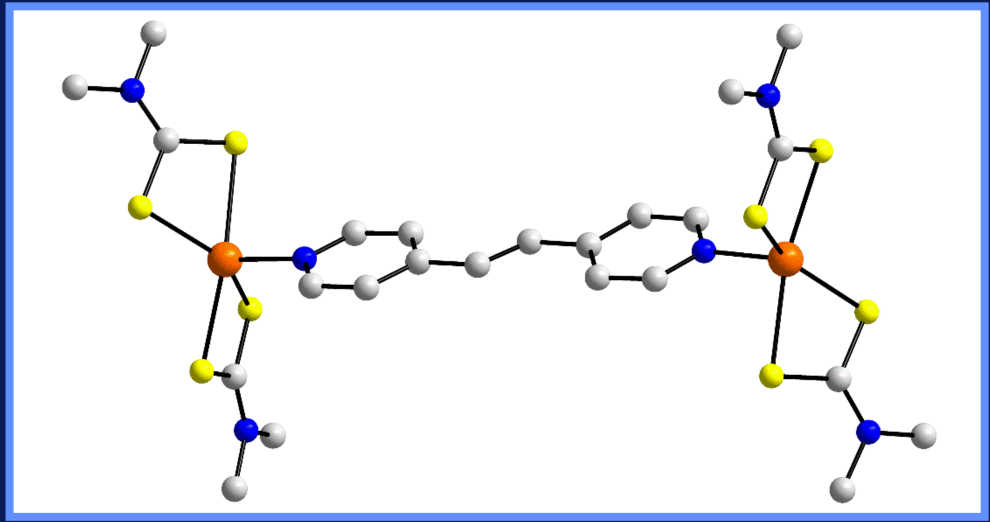


Zinc(dithiocarbamate)$_2$ + bpe

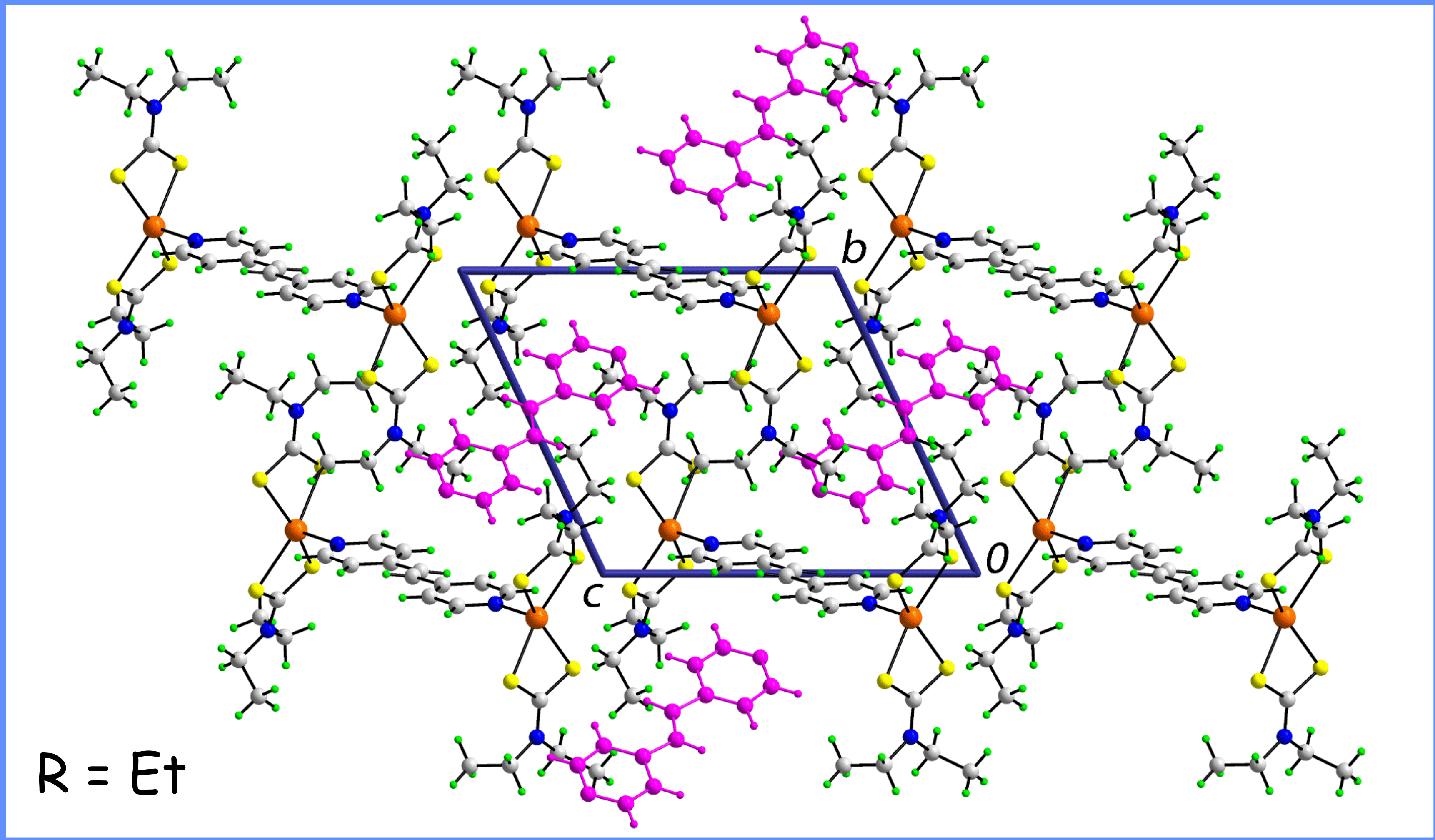
R = Me

R = Et

R = iPr

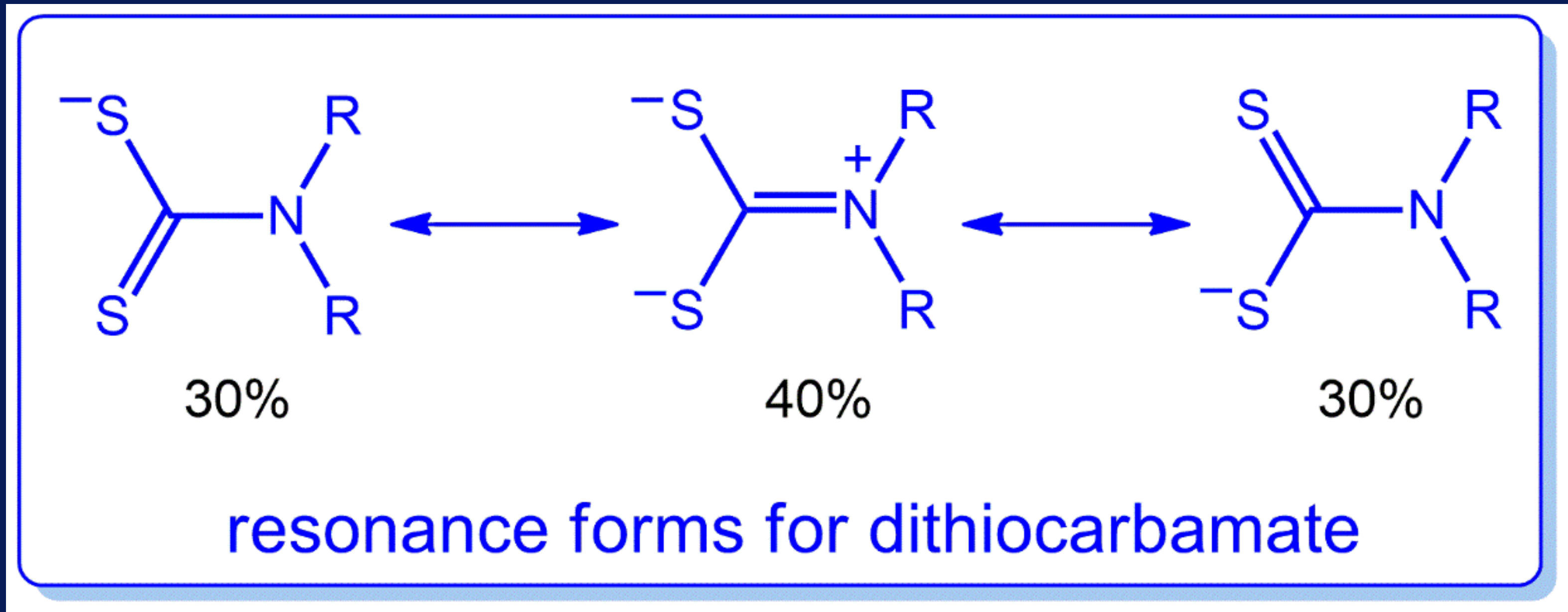


Zinc(dithiocarbamate)$_2$ + bpe



Crystallisation with an excess bpe leads to a lattice adduct

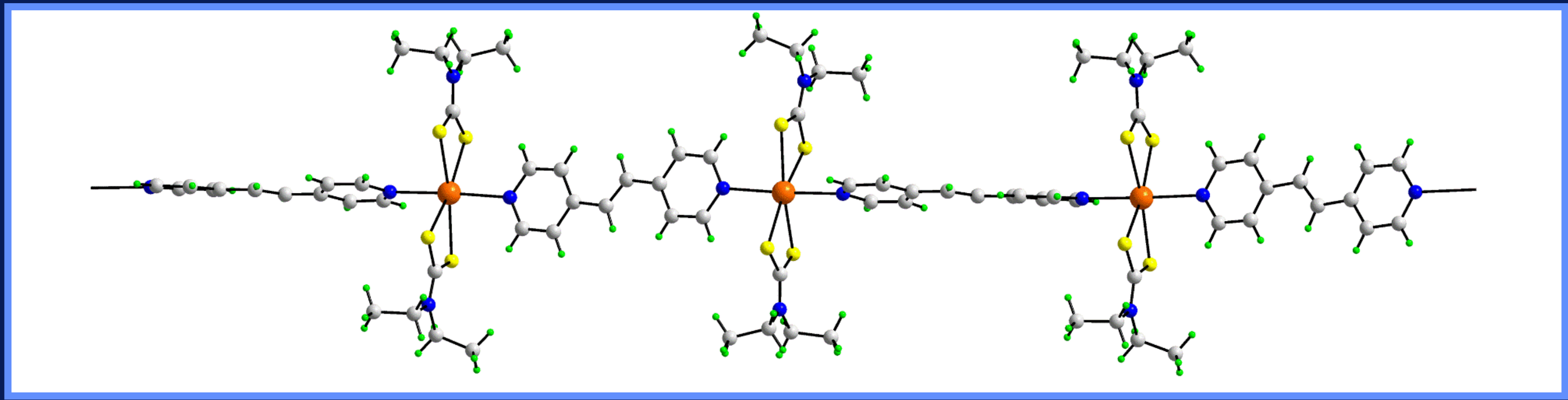
Explanation: electronic effects



Effective chelator for metals and reduces Lewis acidity

Cadmium(dithiocarbamate)$_2$ + bpe # 1

R = Et



Increase the size of the metal centre

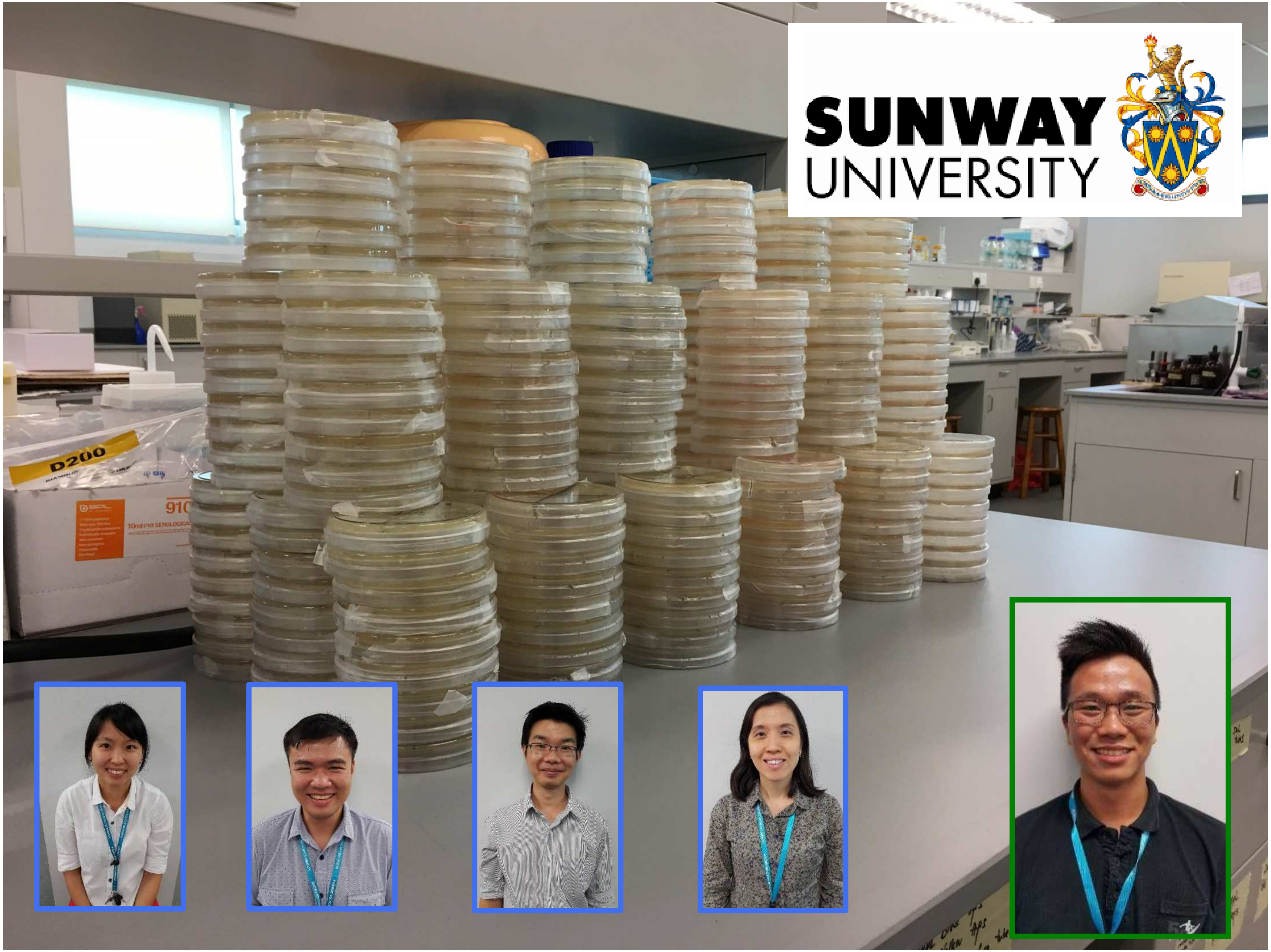
Conclusion #2

One can control supramolecular aggregation in metal 1,1-dithiolates by:

electronic effects

size of the central element

Au···Au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Fortuna Eruditis Favet ("*Fortune favours the prepared mind*")

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