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Gold(I)-selenolate complexes: Synthesis, characterization and ligand exchange reactions

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Abstract. In this paper, the synthesis and characterization of some imidazole-based gold-selenolates are described. This study indicates that the nature of selenolate plays an important role in ligand exchange reactions in gold(I) selenolates. Furthermore, the reactivity of imidazole-based gold(I) selenolates toward nucleophiles such as selenols and phosphines is strikingly different from that of the *N,N*-dimethylaminobenzylamine-based gold(I) complexes. The presence of Se$\cdots$N non-bonded interactions in *N,N*-dimethylaminobenzylamine-based gold(I) complexes modulates the reactivity of Au(I) centre towards incoming nucleophiles.

Keywords. Ligand exchange; gold; phosphines; selenium; selenoenzymes.

1. Introduction

Gold(I) complexes such as auranofin (**1**, AUR), gold thioglucose (**2**, GTG) and gold thiomalate (**3**, GTM) (figure 1) have been used as therapeutic drugs for Rheumatoid Arthritis (RA) for a long period of time.[1] The biochemical mechanism for the action of these compounds in RA is complex as these compounds interact with several biomolecules. Owing to the higher affinity of gold(I) towards sulfur and selenium, gold(I) drugs rapidly react with activated cysteine or selenocysteine residues of enzymes to form protein-gold(I)-thiolate or protein-gold(I)-selenolate complexes.[2] It is known that the gold(I) drugs rapidly bind to the most abundant plasma protein serum albumin (Alb-SH) after their administration, which is important for the transport of these drugs.[3] The interaction of AUR with Alb-SH has been studied extensively in recent years and these studies reveal that AUR reacts with the active site cysteine residue (Cys-34) of Alb-SH to produce the corresponding albumin-gold(I)-phosphine (Alb-S-Au-PEt_3) complex.[3]

Similarly, the gold–phosphole complex **4** has been shown to react with the Cys-284 of human glutathione reductase (hGR) to produce a stable protein-gold(I)-phosphole (Cys284-S-Au-phosphole) complex.[4] Interestingly, gold(I) drugs effectively inhibit several selenoenzymes such as glutathione peroxidase (GPx),[5] thioredoxin reductase (TrxR)[6] and iodothyronine deiodinase (ID-1),[7] by forming gold(I)–selenolate complexes with the selenocysteine (Sec) residues at the active sites. The formation of gold(I)–selenolate complexes in proteins appears to be more facile than the formation of gold(I)–thiolates. For example, the substitution of Sec in ID-1 by a Cys residue significantly reduced the sensitivity of the enzyme toward GTG.[7] Recently, we have shown that trialkyl/aryl gold(I) chlorides (R_3PAuCl, R = Me, Et or Ph) inhibit the GPx activity of the selenol **5** by forming gold(I)-selenolate complexes (**6–8**).[8] Gimeno and co-workers reported similar gold–selenolates (**9–10**) bearing the *N,N*-dimethylbenzylamine moiety (figure 2).[9]

It has been reported that the amino group in **5** plays an important role in the GPx activity as the Se$\cdots$N non-covalent interactions in the key catalytic intermediates modulate the catalytic activity.[8,10] In the natural GPx enzyme, two amino acid residues, glutamine (Gln) and tryptophan (Trp), form a 'catalytic triad' with the Sec residue at the active site.[11] In ID-1 and TrxR, histidine residues (His) have been shown to play crucial roles in the catalysis.[12] However, it is not known whether these proximal amino acid residues modulate the reactivity or stability of the gold–selenolate complexes during inhibition by gold(I) complexes. In this paper, we describe, for the first time, the mode of ligand exchange reactions at Au(I) center in gold–selenolates in the presence or absence of any Se$\cdots$N interactions.

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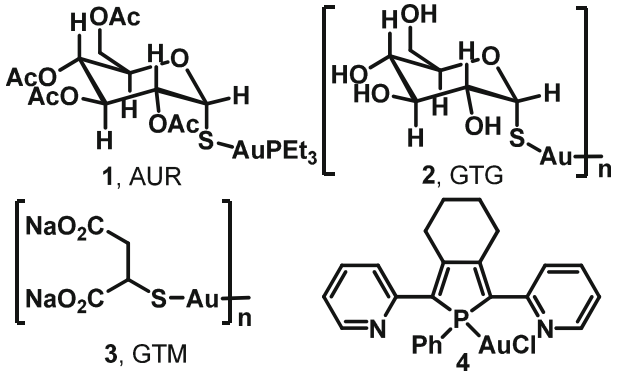


Figure 1. Chemical structures of therapeutic gold(I) compounds **1**–**4**.

2. Experimental

2.1 *General procedure*

Trimethylphosphine gold(I) chloride, triethylphosphine gold(I) chloride, triphenylphosphine gold(I) chloride and sodium borohydride were purchased from Sigma-Aldrich. The experiments were carried out under dry and oxygen-free nitrogen using standard Schlenk techniques for the synthesis. ^{1}H (400 MHz), ^{13}C (100 MHz), ^{31}P (162 MHz) and ^{77}Se (76 MHz) NMR spectra were obtained on a Bruker 400 MHz NMR spectrometer. Chemical shifts are cited with respect to Me_4Si as internal (^{1}H and ^{13}C), H_3PO_4 (^{31}P) and Me_2Se as external (^{77}Se) standards. Mass spectral studies were carried out on a Bruker Daltonics Esquire 6000plus mass spectrometer with ESI-MS mode analysis. The diselenide **11** was synthesized following the literature method.[13a]

2.2 *Synthesis of gold–selenolates **13–15***

To a deoxygenated aqueous solution of the diselenide **11** (30.0 mg, 0.094 mmol) was added sodium borohydride (35.0 mg, 0.940 mmol) to generate the corresponding selenol **12**. The resulting selenol was extracted with deoxygenated chloroform and then the appropriate phosphine gold(I) chloride (0.094 mmol) was added. The reaction mixture was stirred at room temperature for 1 h. The solvent was evaporated *in vacuo*. The gold(I) selenolates **13–15** were obtained as yellow semi-solid products in almost quantitative yields.

2.2a *Compound **13***: ^{1}H NMR ($CDCl_3$) δ (ppm): 1.66–1.69 (d, J = 12.0 Hz, 9H), 3.63 (s, 3H), 6.94 (s, 1H), 7.09 (s, 1H). ^{13}C NMR ($CDCl_3$) δ (ppm): 14.9 (d, $J_{C\text{-}P}$ = 37.0 Hz), 35.1, 118.1, 121.0, 139.9. ^{31}P NMR ($CDCl_3$) δ (ppm): −0.6. ^{77}Se NMR ($CDCl_3$) δ (ppm): 34. ESI-MS m/z calcd. for $C_7H_{14}N_2AuPSe$ $[M+H]^+$ 434.9; found 434.8.

2.2b *Compound **14***: ^{1}H NMR ($CDCl_3$) δ (ppm): 1.10–1.19 (m, 9H), 1.91 (m, 6H), 3.64 (s, 3H), 6.97 (d, J = 2.0 Hz, 1H), 7.09 (d, J = 2.0 Hz, 1H). ^{13}C NMR ($CDCl_3$) δ (ppm): 9.4, 17.9 (d, $J_{C\text{-}P}$ = 34.0 Hz), 36.6, 119.7, 122.5, 141.0. ^{31}P NMR ($CDCl_3$) δ (ppm): 36.1.^{77}Se NMR ($CDCl_3$) δ (ppm): 46. ESI-MS m/z calcd. for $C_{10}H_{20}N_2AuPSe$ $[M+H]^+$ 477.0; found 477.0.

2.2c *Compound **15***: ^{1}H NMR ($CDCl_3$) δ (ppm): 3.68 (s, 3H), 6.88 (s, 1H), 7.10 (s, 1H), 7.42–7.49 (m, 10H), 7.57–7.63 (m, 5H). ^{13}C NMR ($CDCl_3$) δ (ppm): 36.6, 119.5, 121.9, 129.5 (d, $J_{C\text{-}P}$ = 11.0 Hz), 130.0, 130.7, 131.9, 134.7 (d, $J_{C\text{-}P}$ = 13.0 Hz), 143.7, 143.9. ^{31}P NMR ($CDCl_3$) δ (ppm): 35.6. ^{77}Se NMR ($CDCl_3$) δ (ppm): 22. ESI-MS m/z calcd. for $C_{22}H_{20}N_2AuPSe$ $[M+H]^+$ 621.0; found 620.9.

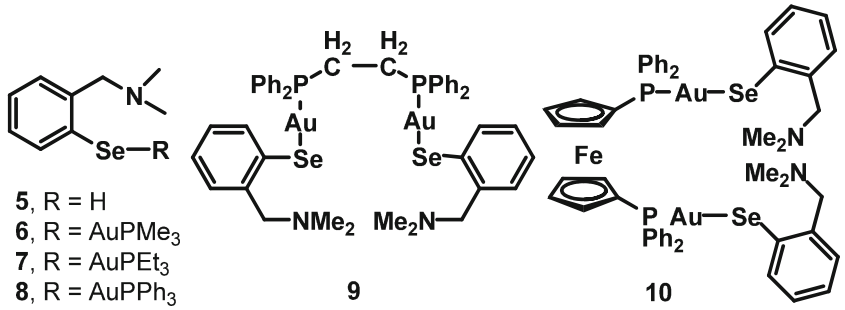


Figure 2. Chemical structures of selenol **5** and gold(I)–selenolate complexes **6**–**10**.

2.3 *General procedure for the reaction of gold–selenolates with selenolate and phosphines*

The reactions of gold–selenolates **13–15** with selenolate **12** and triphenylphosphine (PPh_3) were carried out in NMR tube using MeOH as solvent. To a methanolic solution of complexes **13–15**, was added required amount of either selenolate **12** (freshly prepared by the reduction of the corresponding diselenide **11**) or PPh_3. The resultant solution was mixed thoroughly and then studied by ^{77}Se, ^{31}P NMR and ESI-MS techniques to understand the ligand exchange reactions and to analyse the final products.

2.4 *X-ray crystallography*

Single crystal X-ray diffraction data was collected on a Bruker AXS SMART APEX CCD diffractometer at room temperature (291 K). The X-ray generator was operated at 50 KV and 35 mA using Mo–Kα radiation (λ = 0.71073 Å). The data was collected using SMART software package.[14] The data were reduced by SAINTPLUS,[14] an empirical absorption correction was applied using the package SADABS[15] and XPREP[14] was used to determine the space group. The crystal structure was solved by direct methods using SIR92[16] and refined by full-matrix least-squares method using SHELXL97.[17] All non-hydrogen atoms were refined anisotropically and hydrogen atoms were assigned at idealized locations.

2.4a *Crystal data for compound **13***:[18] $C_7H_{15}N_2PSeAuCl$; M_r = 469.5; monoclinic; space group: $P21/c$; a = 11.8096(28) Å; b = 26.4283(61) Å; c = 11.5510(27) Å; α = 90.00(0)°; β = 113.270(4)°; γ = 90.000(0)°; V = 3311.89(63) Å^3; ρ_{calc} = 1.88 g m^{-3}; Z = 8; MoK_α radiation (λ = 0.71370 Å); T = 291(2) K; R_{int}: 0.054; R (observed data): R_1 = 0.061; wR_2 = 0.105; R (all data): R_1 = 0.043; wR_2 = 0.099; GOF = 0.929; $\Delta\rho_{min}$ and $\Delta\rho_{max}$ (eÅ^{-3}): −1.733 and 2.010.

3. Results and discussion

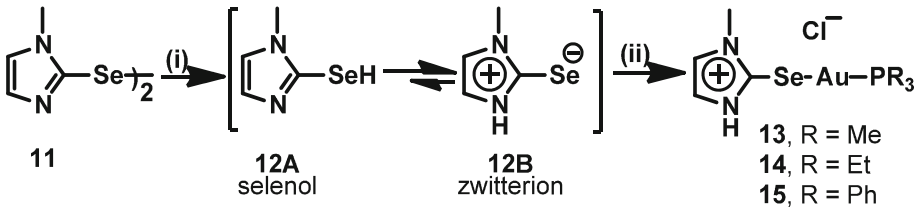
3.1 *Synthesis of gold(I)–selenolates*

The imidazole-based gold–selenolates **13–15** were synthesized by treating R_3PAuCl (R = Me, Et or Ph) with the selenolate **12** (scheme 1). The diselenide **11** can be quantitatively reduced to the corresponding selenol **12** by the treatment of $NaBH_4$.[13] The selenol produced in this reaction exists predominantly in its zwitterionic form (**2B**) having a large negative charge on selenium atom as shown in scheme 1. A nucleophilic attack of the selenol **12** at the Au(I) centre of R_3PAuCl produces the corresponding gold–selenolates **13–15** almost in quantitative yields. All the gold–selenolates were characterized by NMR (^1H, ^{13}C, ^{31}P and ^{77}Se) spectroscopic and ESI-MS spectrometric techniques.

3.2 *Structural features of gold(I)–selenolate **13***

To understand the important structural features in the gold–selenolates, complex **13** was studied by single crystal X-ray diffraction. After the synthesis of complex **13** in chloroform, the crude compound was recrystallized from chloroform/methanol (1:1) mixture to afford faint yellowish needle-shaped crystals. As shown in figure 3, the structure of complex **13** was found to be dimeric with a weak Au···Au non-covalent aurophilic interaction (dAu1···Au2 = 3.104 Å), which was much shorter than the sum of van der Waals radii of Au atoms (3.40 Å).

As expected, an almost linear arrangement of Se–Au–P moieties were observed (θSe1-Au1-P1: 175.9° and θSe2-Au2-P2: 172.4°), which is a characteristic of Au(I) complexes.[4,9] A small deviation from the linearity is probably due to the presence of Au···Au aurophilic interaction. Similar structural features were also observed for complexes **9** and **10** as reported by Gimeno and co-workers.[9] While the Au···Au distance of 3.104 Å in complex **13** is comparable to that of **9** (3.091 Å), the distance is found to be slightly longer



Scheme 1. Synthetic routes to the gold–selenolates (**13–15**). (i) $NaBH_4$, $CHCl_3$/H_2O; (ii) R_3PAuCl (R = Me, Et or Ph), $CHCl_3$.

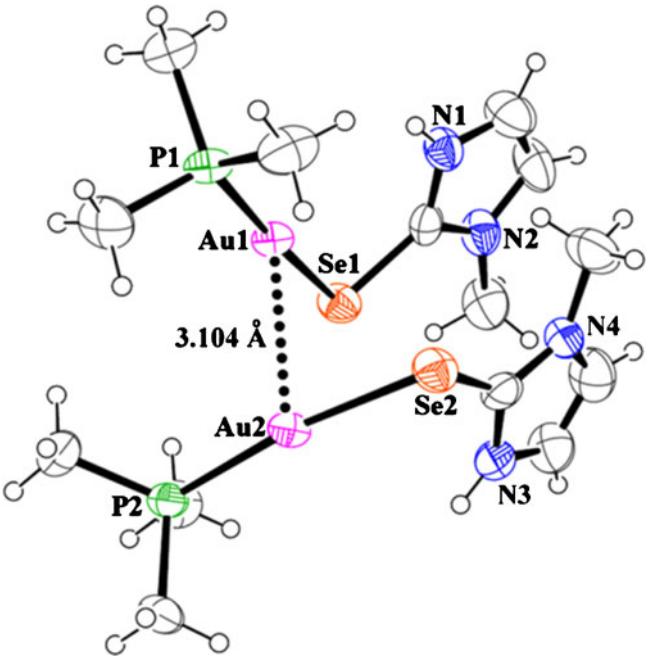


Figure 3. X-ray crystal structure of gold–selenolate complex **13**. Displacement ellipsoids are drawn at 50% probability level and hydrogen atoms are shown as small spheres of arbitrary radii. Two chloride counterions (Cl^-) in the molecule are omitted for the structural clarity.

than that reported for complex **10** (3.024 Å). Although two Cl^- counterions were present in the crystal structure of complex **13**, both of the Cl^- ions were far away from the Au and Se centres. Furthermore, both the Cl^- ions were located nearer to one imidazole-gold-selenolate unit. For example, the distance of Cl^- ions from Au2 and Se2 are found to be much higher than the distance from Au1 and Se1 centres. However, all these distances are much higher than the sum of van der Waals distance of Se/Au and Cl atoms indicating the absence of any non-bonded interactions (table 1).

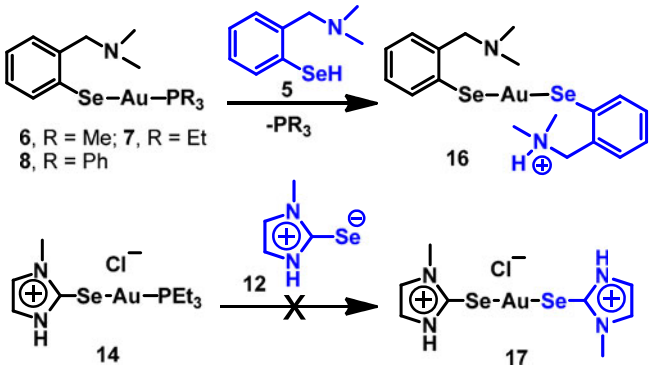
3.3 *Ligand exchange reactions in gold(I)-selenolates*

It has been shown that ligand exchange reactions play an important role in the inhibition of GPx.[5] For example, GTG undergoes ligand displacement reactions with glutathione (GSH) to produce $[Au(SG)_2]^-$ complex, which is probably responsible for the inhibition of the enzyme.[5c] The synthetic gold–selenolate complexes **6–8** also undergo extensive ligand exchange reactions in the presence of selenol **5** and phosphines.[8] In these reactions, the trialkyl/aryl phosphines were eliminated, which underwent further reactions with oxygen and selenium powder to produce the corresponding phosphine oxides ($R_3P{=}O$) and selenides ($R_3P{=}Se$).[8] In contrast, reactions of the imidazole-based selenol **12** with trialkyl/aryl gold chlorides produced the corresponding gold–selenolates (**13–15**) and the formation of phosphine oxides or selenides was not observed. These observations indicate that the Se···N interactions in complexes **6–8** may activate the S–Au and Au–P bonds toward ligand displacement reactions. When complex **6** was treated with **5**, the formation of *bis*-selenolate–gold complex **16** was observed due to the replacement of the phosphine ligand by the selenol (scheme 2).[8] In contrast, no such reaction was observed when complex **14** was treated with an equimolar amount of compound **12**. Although a small amount of triethyl phosphine oxide ($Et_3P{=}O$) was observed in the reaction, the ESI-MS spectrum indicated that the selenolate and phosphine ligands are intact in complex **14** (figures S13 and S14 of Supporting Information). These observations indicate that the notable differences in reactivity of complexes **6–8** and **14** towards corresponding selenols are probably due to the differences in nucleophilicity/basicity of $–NMe_2$ and Cl^- moieties.

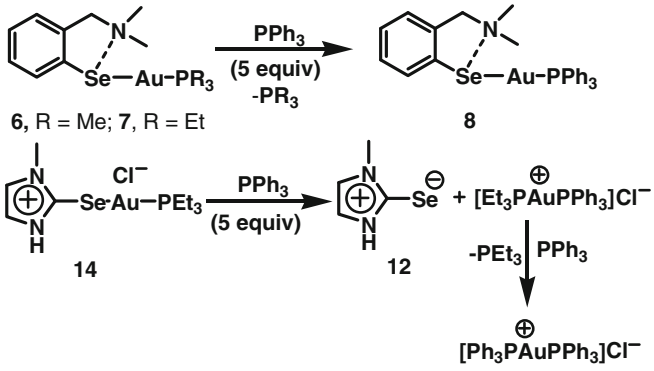
A striking difference in the reactivity of these two types of gold–selenolates toward selenols prompted us to investigate the phosphine exchange reactions. As

Table 1. Some important bond distances, bond angles and non-bonding distances in the crystal structure of complex **13**.

Au1–Se1	2.440(1)	Au2–Au1–Se1	83.33(3)	Au1···Au2	3.104
Au1–P1	2.267(2)	Au2–Au1–P1	102.72(6)	Se1···Cl1	4.926
Au2–Se2	2.4396(9)	Se1–Au1–P1	172.41(7)	Se1···Cl2	8.988
Au2–P2	2.266(2)	Au1–Au2–Se2	86.89(2)	Au1···Cl1	3.947
Se2–C8	1.882(9)	Au1–Au2–P2	96.97(6)	Au1···Cl2	6.620
Se1–C1	1.89(1)	Se2–Au2–P2	175.98(7)	P1···Cl1	4.002
P1–C6	1.79(1)	Au2–Se2–C8	101.1(3)	P1···Cl2	4.441
P1–C5	1.80(1)	Au1–Se1–C1	96.8(3)	Se1···Au2	3.719
P1–C7	1.79(1)	Au1–P1–C6	110.5(4)	Se2···Au1	3.842
P2–C13	1.83(1)	Au1–P1–C5	112.8(4)	N1H1···Cl1	2.298
P2–C12	1.82(1)	Au1–P1–C7	116.5(4)	N1···Cl1	3.109



Scheme 2. Nucleophilic attack of the selenol **5** at the Au(I)-centre in gold–selenolates **6**–**8** produces the bis-selenolate-gold complex **16**. No such reaction was observed when the selenolate **12** was added to complex **14**.

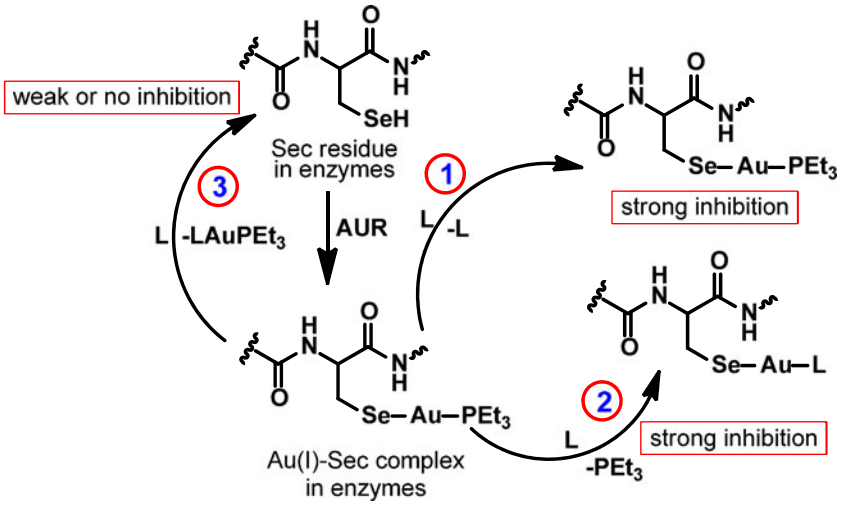


Scheme 3. Differences in the reactivity of complexes **6**, **7** and **14** towards PPh_3.

shown previously, addition of PPh_3 to the benzylamine-based gold–selenolate complexes **6**–**7** leads to a rapid elimination of PMe_3 or PEt_3 to produce complex **8**.[8] In contrast, the imidazole-based gold–selenolates **13**–**14** do not undergo any phosphine displacement reactions in the presence of PPh_3. For example, the treatment of **14** with an excess amount (~5 equiv) of PPh_3 does not produce the phosphine-exchanged product **15**. Interestingly, the ^{31}P NMR spectroscopic and ESI-MS spectrometric analyses revealed the formation of $[Et_3PAuPPh_3]^+$ species, indicating that the addition of PPh_3 led to the elimination of the selenolate ligand. The $[Et_3PAuPPh_3]^+$ species produced in the reaction reacts with PPh_3 to produce $[(PPh_3)_2Au]^+$ cation by phosphine exchange reactions (scheme 3). Furthermore, the produced cationic gold–phosphine complexes such as $[Et_3PAuPPh_3]^+$ and $[(PPh_3)_2Au]^+$ may undergo a charge balance with Cl^- counterions as shown in scheme 3. As expected, a line broadening in ^{31}P NMR signal was observed during the course of phosphine exchange reactions. Although there is no direct evidence, the elimination of selenolate in the reaction of **14** with PPh_3 is probably due to the stronger *trans* effect of the phosphine ligand as compared to the selenolate ligand, which leads to the weakening of the Au–Se bond. Whereas, the Se···N interactions in the benzylamine-based gold–selenolates **6**–**8** may reduce the *trans* directing effect of phosphine group, leading to the weakening of the Au–P bonds. This may facilitate the phosphine exchange reactions as shown in scheme 3. These observations lead to the assumption that the non-bonded interactions at the selenium centres in gold–selenolates may modulate the ligand exchange reactions at the Au(I) centres.

3.4 *Possible pathways of different ligand exchange reactions*

Coffer *et al.* have previously shown that the nucleophilic attack of the Cys-34 residue of albumin at the Au(I) centre of AUR leads to the formation of protein–gold–phosphine (Alb–S–$AuPEt_3$) complex, indicating that the replacement of thioglucose moiety is more favoured than that of the PEt_3 ligand.[3a] However, it is not clear whether the elimination of gold(I)–phosphine moiety from an enzyme active site alters the inhibition property of the gold(I) complexes. The present study indicates that the nature of selenolate can alter the ligand exchange reactions at the gold(I) centre. This study also suggests that the effect of gold(I) drugs on selenoenzymes not only depends on the reactivity of Sec toward these gold(I) complexes, but also the stability of Se–Au bond in the enzyme-bound complex. For example, the presence of a ligand (L) in biological medium may have different effects on the gold(I)–selenocysteine complexes as shown in scheme 4. In the first scenario, the addition of L does not lead to any ligand displacement reactions. In such case, the enzyme activity is expected to be strongly inhibited by the treatment of enzyme with gold(I) complexes such as AUR. In the second pathway, the replacement of phosphine ligand by L may not affect the inhibition as the Sec residue is still bonded to the gold(I) moiety. In contrast, a recovery of enzyme activity is expected when the ligand L attacks at the Au(I) centre to release the catalytically active selenol (scheme 4). Therefore, the ligand exchange reactions at the Au(I) centres in protein–gold complexes may or may not affect the inhibition of enzymatic activity depending on the relative strength of protein(Sec)–gold and gold–phosphine bonds. Furthermore, this scheme indicates that, for an effective



Scheme 4. Schematic representation of different ligand exchange reaction pathways in gold(I)–selenocysteine complexes in enzymes.

inhibition of selenoenzymes by gold(I) drugs, the Se–Au bond should be more stable than that of the Au–P bonds.

4. Conclusion

In summary, some imidazole-based gold(I)–selenolates have been synthesized, characterized and studied for their reactivity towards different nucleophiles. This study indicates that the nature of selenolate plays an important role in ligand exchange reactions in gold–selenolates. The reactivity of the imidazole-based gold(I)–selenolates toward selenols and phosphines is significantly different from that of the *N,N*-dimethylbenzylamine-based gold(I) complexes. The strong *trans* effect of phosphines probably makes the Au(I)–Se bond relatively weaker, leading to the cleavage of Au(I)–Se bond in gold(I)–selenolates upon the nucleophilic attack of selenols and phosphines. These observations suggest that the effect of gold(I) drugs on selenoenzymes may not only depend on the reactivity of Sec toward these gold(I) complexes, but also on the stability of Se–Au bond in the gold(I)–selenolate complex of the enzyme.

Supporting information

NMR and ESI-MS spectral data of all the gold–selenolate complexes are given in supplementary information (figures S1–S18). Supplementary data to this article can be found in the Website www.ias.ac.in/chemsci.

Acknowledgements

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18. CCDC-828285 (**13**) contains the supplementary crystallographic data for this paper. These data can be obtained free of charge from The Cambridge Crystallographic Data Centre *via* www.ccdc.cam.ac.uk/data_request/cif