the product is obtained with 87.8% after only 4 min of hydrogenation at normal pressure at 25 °C.[16] The lower activity in toluene is a result of the "induction periods"[7] as well as the formation of stable, blocking arene complexes, as we have now shown quantitatively.[17]

In summary, we have shown that arene complexes of Rh^I can have an unexpectedly large influence on the activity of asymmetric hydrogenation reactions. A different interpretation is now possible for past investigations in which catalyst activities were determined in alcohol/aromatic solvent mixtures or in aromatic solvents.[18] At the moment, we are examining whether the inhibiting effect can also be induced by P-ligands and by substrates with aromatic substituents, and whether it is also relevant for other Rh^I-catalyzed reactions, for example, hydroformylations.

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The Core Structure of TMC-95A Is a Promising Lead for Reversible Proteasome Inhibition**

Markus Kaiser, Michael Groll, Christian Renner, Robert Huber, and Luis Moroder*

The proteasome is an intracellular multicatalytic protease complex which in combination with the ubiquitin pathway plays a central role in major cellular processes, such as antigen presentation, cell proliferation and differentiation, and apoptosis.[1] Proteolysis occurs in a barrel-shaped core structure known as 20S proteasome, which consists of four stacked rings arrayed in an $\alpha_7\beta_7\beta_7\alpha_7$ mode.[2a] In eukaryotic proteasome three β-subunits of each β-ring are enzymatically active with an N-terminal threonine residue as the active nucleophile involved in proteolysis[2b] with three more or less distinct substrate specificities, that is chymotrypsin-like (CL), trypsin-like (TL), and peptidyl-glutamyl-peptide hydrolase (PGPH) activities.[3]

Because of the physiological role of proteasome in critical intracellular processes, this enzyme represents a promising target for drug development in inflammatory and autoimmune diseases as well as in tumor therapy.[4] Correspondingly, great attention has recently been paid to the discovery of potent and selective proteasome inhibitors by structure-based design or natural product screening approaches. Most of the synthetic inhibitors consisting of peptide aldehydes, boronates, and vinylsulfones, as well as the natural products lactacystin and epoxymicins inhibit in a more or less selective manner the proteasome by reaction with the N-terminal threonine residue (for a recent review see ref. [5]). A notable exception is the highly selective and competitive proteasome inhibitor TMC-95A, which was isolated from the fermentation broth of *Apiospora montagnei* Sacc. TC 1093.[6]

This cyclic peptide metabolite consists of L-tyrosine, L-asparagine, a highly oxidized L-tryptophane, and the (*Z*)-1-

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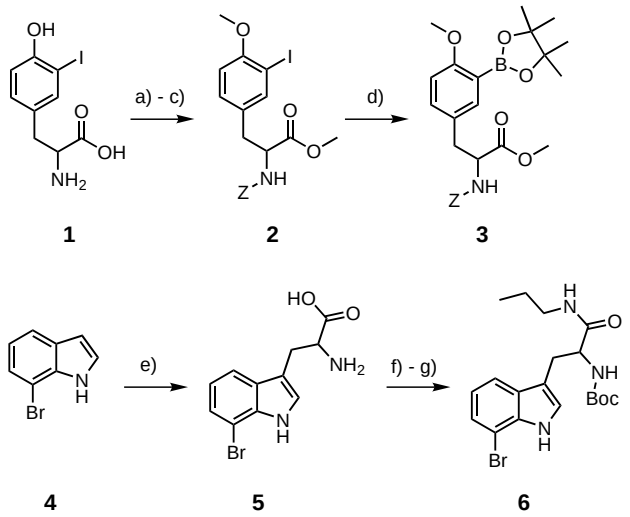
propenylamine and 3-methyloxopentanoic acid moities with a phenyl/oxindole ring junction (structure A in Scheme 1).[7] A similar biaryl moiety was so far encountered only in chloropeptin,[8] complestatin,[9] diazonamide,[10] and the kistamicins.[11] Because of the pharmacological interest of proteasome inhibition and the distinct phenol/oxindole system the synthesis of TMC-95A has attracted considerable interest.[12]

X-ray structural analysis of the complex of TMC-95A with yeast proteasome clearly confirmed the noncovalent binding of this low molecular weight cyclic peptide, with a network of hydrogen bonds between the β-type extended peptide moiety and the main chain of the protein[13] which contributes to the high affinity for all three active sites.[6] From this crystal structure the minimum structural elements of TMC-95A for binding to the proteasome were derived (structure B in Scheme 1). To validate our working assumption we selected compound C (Scheme 1) as our first synthetic target; compound C contains a propylamide residue as R^1 for interaction with the S1 pocket of the active sites, and the side chain of asparagine, which is present in the natural product, as R^3 for occupancy of the S3 pocket. All other modifications at the level of the core structure of TMC-95A were not expected to significantly affect the binding affinity for proteasome. The synthesis of this first TMC-95A analogue was accomplished and its inhibitory potency fully confirmed our working hypothesis.

The main difficulty in the synthesis of compound C was expected from cyclization to the constraint ring structure. Based on previous experiences reported for the synthesis of the chloropeptin ring,[14] a first synthetic route by acid-catalyzed ring contraction of an 18-membered macrolactame precursor was attempted. This foresees a Suzuki cross-reaction for the preparation of the biaryl system followed by lactamization of the linear precursor. As meanwhile also reported by Estiarte et al.,[15] cyclization of the 18-membered precursor failed. Thus, the compound C was synthesized according Scheme 2 and 3 by formation of the "correct" biaryl junction at the level of the linear precursor followed by cyclization.

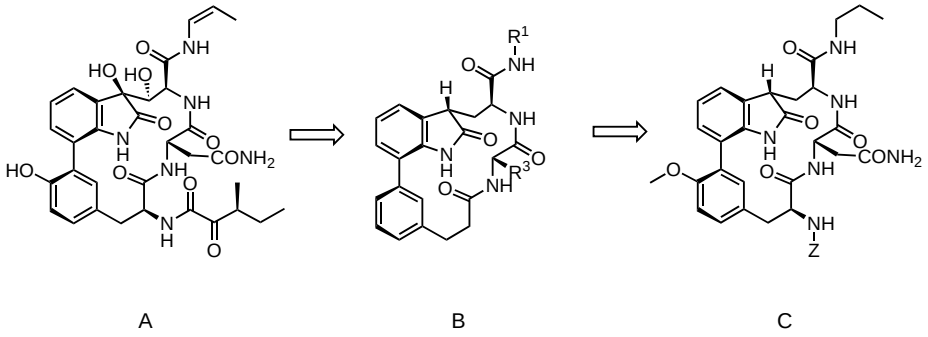
For this synthetic route 3-iodo-L-tyrosine was converted into the suitably protected derivative **2** which was then transformed to the aryl boronate **3** by the Miyauri–Suzuki reaction[16] using bis(pinacolato)diboron, [$PdCl_2$(dppf)], and KOAc (Scheme 2). 7-Bromo-L-tryptophane was produced from 7-bromoindole and L-serine by the use of tryptophane synthetase following essentially known protocols[17] and then



Scheme 2. Synthesis of **3** and **6**: a) $SOCl_2$, MeOH; b) Z-OSu; c) K_2CO_3 (10 equiv), MeI (5 equiv); d) $B_2O_4C_{12}H_{24}$ (1.1 equiv), [Pd(dppf)Cl_2]·CH_2Cl_2 (5 mol%), KOAc (3 equiv); e) L-serine, tryptophane-synthetase, PLP, 37 °C; f) Boc_2O; g) nPrNH$_2$, EDCI, HOBt. Z-OSu = N^{α}-(benzyloxycarbonyloxy)succinimide, dppf = $Ph_2PC_5H_4FeC_5H_4PPh$, PLP = pyridoxal 5′-phosphate, Boc = *tert*-butoxycarbonyl, EDCI = *N*′-(3-dimethylaminopropyl)-*N*-ethylcarbodiimide, HOBt = 1-hydroxy-1*H*-benzotriazole, DME = dimethoxyethane.

converted into the related N^{α}-Boc propylamide derivative **6** (Scheme 2). The derivatives **3** and **6** were coupled under standard conditions of the Suzuki cross-reaction[18] to generate the key intermediate **7** in 80% yield after flash chromatography (Scheme 3). Upon C-terminal extension of the peptide chain with the asparagine *tert*-butyl ester, the linear precursor **8** was oxidized at the indole moiety by standard procedures with DMSO/concentrated HCl[19] (40% yield) prior to peptide cyclization with PyBOP/HOBt/DIEA. After HPLC purification the desired compound C (**10**) was isolated in about 40% yield as a homogeneous product (Scheme 3). NMR analysis confirmed the correct ring structure including the *S* configuration at the C3 atom of the oxindole.[20] Apparently only this configuration allows cyclization of the precursor into the highly restricted ring. From distance and dihedral angle constraints derived from 2D NMR spectra a spatial structure of compound C was calculated which is superimposable to that of TMC-95A determined by X-ray analysis of its complex with the proteasome (Figure 1).

In the context of the structural analysis of the TMC-95A/yeast proteasome complex the natural product was found to inhibit the three proteolytic activites of yeast and



Scheme 1. Structure of TMC-95A (A), the minimal skeleton (B) for binding to the proteasome as derived from the X-ray structure of the TMC-95A/proteasome complex, and the analogue (C) with propylamide as R^1 and the side chain of asparagine as R^3 residue. Compound C was synthesized for the validation of the design concept.

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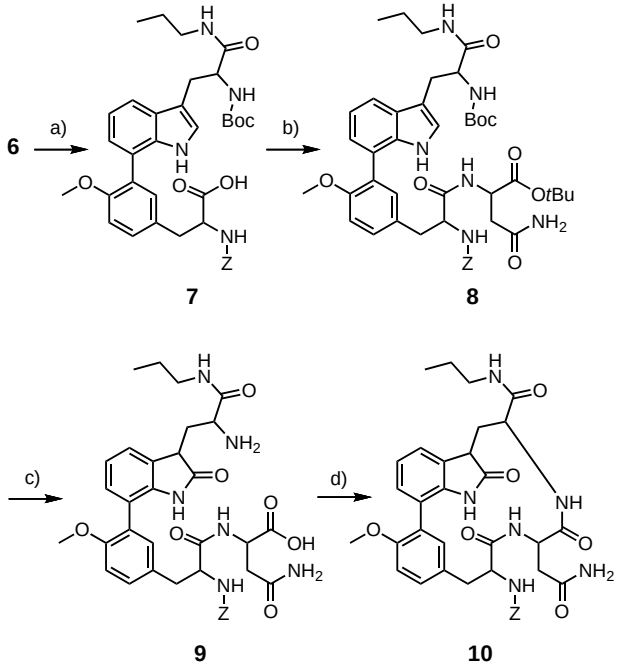

Scheme 3. Synthesis of **10**. a) **3** (1.1 equiv), $[Pd(dppf)Cl_2]\cdot CH_2Cl_2$ (5 mol%), K_2CO_3 (3 equiv), DME/H_2O (7:1), 70 °C; b) H-Asn-O*t*Bu, EDCI, HOBt; c) DMSO (20 equiv), AcOH/HCl (4:1); d) PyBOP (4 equiv), HOBt (4 equiv), DIEA (6 equiv). PyBOP = benzotriazol-1-yloxytripyrrolidinophosphonium hexafluorophosphate, DIEA = diisopropylethylamine.

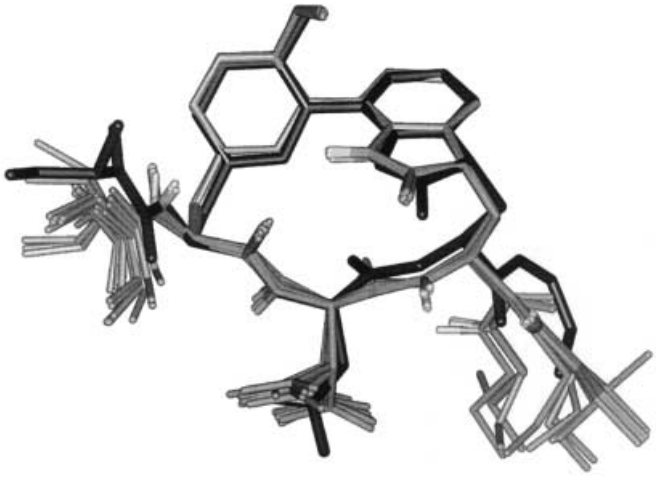

Figure 1. Conformation of compound C determined by NMR spectroscopy (20 lowest energy structures; gray) superimposed on the three-dimensional structure of TMC-95A as determined by X-ray analysis of its complex with yeast proteasome (black); for better visualization the N-terminal phenyl group is omitted.

human proteasome with similar potencies[13] as previously reported for human proteasome.[6] The inhibitory effects of compound C are reported in Table 1, and compared with those of TMC-95A and the calpain inhibitor I, that is Ac-Leu-Leu-Nle-H. Despite the difficult quantitative comparison of IC_{50} values obtained with different proteasome preparations and assay conditions, the synthetic TMC-95A analogue was found to inhibit all three proteolytic activities more efficiently than the tripeptide aldehyde. Compared to the natural product, compound C retains almost full inhibition of the

Table 1. Inhibition of proteasome (IC_{50} [μM]) by compound C, TMC-95A, and Ac-Leu-Leu-Nle-H in absence of SDS.[21]

	CL activity	TL activity	PGPH activity
compound C	8.0[a]	10.6[a]	7.4[a]
	1.9[b]	3.7[b]	1.8[b]
TMC-95A[6]	0.012[b]	1.5[b]	6.7[b]
Ac-Leu-Leu-Nle-H	35.4[a]	142.5[a]	88.0[a]

[a] Yeast proteasome. [b] Human proteasome.

TL and PGPH activities, but it is significantly less potent against the CL activity. This may well be attributed to the exchange of the (*Z*)-1-propenylamide with the more flexible propylamide as P1 residue for occupancy of the specificity subsite S1 of the enzyme. Full retainment of affinity of compound C for the two other active sites of the proteasome confirms that the N-terminal acyl residue as well as the tyrosine hydroxy group are not critically involved in the interaction with the protein counterpart, whilst the biaryl moiety restricts the peptide backbone into the extended β-strand conformation for optimal hydrogen bonding to the active site clefts. The data also confirm that the degree of oxidation of the tryptophane residue can be reduced and that the oxindole group is sufficient for the additional hydrogen bond to the protein backbone to be established.

In conclusion, the inhibitory potencies of compound C confirm the correctness of our inhibitor design based on minimization of the TMC-95A skeleton, and clearly indicate that by optimization of the R^1 and R^3 residues both selectivity and affinity for the three active sites may significantly be improved. These results also suggest that not all of the complex structural elements of the natural product are required for inhibition of proteasome, thus markedly facilitating the synthesis of TMC-95A related compounds.

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[20] NMR experiments were performed at 22 °C in [D_6]DMSO at 500 MHz (Bruker DRX500 spectrometer). 2D-^{1}H DQF-COSY, TOCSY (70 ms mixing) and ROESY (200 ms mixing) spectra were acquired for resonance assignment and conformational analysis. Forty five distance constraints and three Φ dihedral angle constraints were extracted from ROESY and 1D-^{1}H spectra, respectively. A total of 100 independent structures were generated by a distance geometry algorithm, refined by two steps with a simulated annealing molecular dynamics (MD-SA; coarse + fine) protocol and sorted according to final energies. No significant violations of experimental constraints occurred for any of the calculated structures.
[21] The IC_{50} values for yeast and human 20S proteasome were determined at 37 °C in the absence of SDS using the fluorogenic substrates Suc-Leu-Leu-Val-Tyr-AMC, Bz-Thr-Val-Arg-AMC and Z-Leu-Leu-Glu-βNa to assay the CT, TL, and PGPH activities following essentially protocols reported previously (G. Loidl, H.-J. Musiol, M. Groll, R. Huber, L. Moroder, *J. Pept. Sci.* **2000**, *6*, 36 – 46).

P_2-Ligand Complexes as Building Blocks for the Formation of One-Dimensional Polymers**

Junfeng Bai, Eva Leiner, and Manfred Scheer*

Dedicated to Professor Eckhard Herrmann on the occasion of his 65th birthday

Self-organisation of discrete units to form supramolecular aggregates and networks of inorganic coordination compounds is a prominent field in contemporary chemistry.[1] Within this field coordination polymers are of interest with regard to their physical, electronic, catalytic, and structural properties.[2] Usual approaches in this area make use of N-donor containing ligands and heterocycles to connect different metal centers together. However, our goal in this field is to use P_n ligand complexes as connecting moieties between metal cations to form well-oriented assemblies as well as one-dimensional (1D) and two-dimensional (2D) polymers.

The coordination chemistry of P_n ligand complexes[3] towards cationic metal centers (excluding cationic organometallic complex moieties[4]) has to date been limited to the use of *cyclo*-P_3 ligand complexes, such as [(triphos)M(η^3-P_3)] (M = Co, Rh, or Ir; triphos = 1,1,1-tris(diphenylphosphanylmethyl)ethane), which react with Cu^I, Ag^I, or Au^I to form metal-bridged dimers.[5] In the reaction of the Co compound with CuBr, a multidecker complex of Co containing a $(CuBr)_6$ middle deck was obtained.[6] To generate definite polymers, P_2-ligand complexes seem to be the starting material of choice. From our experience with the chromium complex [{$CpCr(CO)_2$}$_2$(μ,η^2-P_2)][7] (Cp = C_5H_5) we know that this starting material readily undergoes fragmentation reactions;[8] thus, we decided to use the more stable Mo analogue **1**.[9] Herein we report the synthesis of a molecular cationic complex of Ag^I containing different and novel coordination modes of the P_2-ligand complex as well as the formation of the first 1D chain polymers of Ag^I and Cu^I containing P_2-ligand complexes.

[$Cp_2Mo_2(CO)_4$(μ,η^2-P_2)] **1**

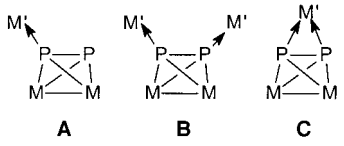
[Ag_2⟨{$Cp_2Mo_2(CO)_4$(μ,η^2:η^2-P_2)}$_2$⟩ ⟨{$Cp_2Mo_2(CO)_4$(μ,η^2:η^1:η^1-P_2)}$_2$⟩][(CF_3SO_3)$_2$] **2**

[{$Re(CO)_3Br$}$_2$⟨{$Cp_2Mo_2(CO)_4$(μ,η^2:η^1:η^1-P_2)}$_2$⟩] **3**

[Ag_2{$Cp_2Mo_2(CO)_4$(μ,η^2:η^1:η^1-P_2)}$_3$(μ,η^1:η^1-NO_3)]$_\infty$[NO_3]$_n$ **4**

[Cu(μ-Br){$Cp_2Mo_2(CO)_4$(μ,η^2:η^1:η^1-P_2)}]$_\infty$ **5**

The reaction of $Ag(CF_3SO_3)$ with **1** in CH_3CN leads to the quantitative formation of **2**, in which the tetrahedral Mo_2P_2 ligand coordinates to two Ag(I) centers in a bridging as well as in a novel chelating coordination mode. Whereas the coordination of M_2P_2 tetrahedral complexes to one (type **A**) and two (type **B**) organometallic moieties is known, such as, in the type **B** situation where coordination to [$Re(CO)_3Br$] fragments forms a central six-membered ring moiety in complex **3**,[10] the side-on coordination mode of an M_2P_2 complex at one metal center (type **C**), as found in complex **2**, is unique for a P_2-ligand complex.[3]



If the counterions at the Ag^I centers are noncoordinating, complexes similar to **2** are obtained.[11] However, we found a different coordination behavior if the counterion is incorporated into the coordination sphere of the Ag^I atom. Thus, the reaction of **1** with $AgNO_3$ leads to the quantitative formation of a novel polymeric Ag^I compound **4**, where one of the NO_3 counterions is now incorporated into the coordination sphere of the Ag^I atoms to form a waved 1D polymeric chain.

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