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ORIGINAL PAPER

Oxidation of 3,5-di-*tert*-butylcatechol in the presence of V-polyoxometalate

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The oxidase and dioxygenase reactions of 3,5-di-*tert*-butylcatechol (DTBC, *I*) in the presence of V-polyoxometalate were studied. It was found that the addition of a Lewis base quenched the V-polyoxometalate-catalysed catechol dioxygenase reaction and catalysed the oxidase reaction selectively. The existence of V-polyoxometalate accelerates the autoxidation rate of *I* as demonstrated by the rate measurements. ESR and UV-VIS spectra showed that the Lewis base destroyed the dioxygenation reaction catalyst as formed and restrained its regeneration by suppressing the coordination of catechol radical to vanadium. The by-products of the dioxygenation and oxidation reactions are H_2O and H_2O_2, respectively.

Keywords: 3,5-di-*tert*-butylcatechol, catechol oxidase, base-catalysed, V-polyoxometalate-enhanced

Introduction

The oxidation of organic substrates and degradation of aromatic organic pollutants using molecular oxygen are of great interest, from both the economic and environmental perspectives. Metal-containing redox enzymes are ubiquitous in nature and many can perform such functions under mild conditions (Sigel & Pyle, 2007; Cowan, 1998; Ragsdale & Kumar, 1996). Catechol oxidase and catechol dioxygenases are two important enzymes in these investigations. Oxygenases catalyse the incorporation of oxygen atoms from molecular oxygen into the substrate, while oxidases use dioxygen as an electron acceptor. Many functional model complexes for metalloenzymes have been studied extensively in order to elucidate the reaction mechanisms involved and to help design biomimetic catalyst systems for various reactions requiring activation of O_2 (Lin et al., 2001; Cox & Que, 1988; Koval et al., 2006; Gao & Xu, 2006). Morris et al. (2009) reported the vanadium-catalysed dioxygenase reaction of catechol by activating oxygen. They found that the vanadium-based catechol dioxygenation was initiated via the oxidase product: quinone. The induction period which preceded the dioxygenation reaction was the time for the formation of quinone and then the real dioxygenase catalyst (Yin et al., 2005). The oxidase-product-induced catalysis was thought to be general and had been verified by independent examples of both oxidative and reductive catalysis (Hagen et al., 2005; Groves et al., 1996). Research into the oxidase reaction mechanism and the relationship between the oxidase reaction and dioxygenation reaction are not only beneficial in elucidating the mechanism of the underlying biological catecholase reaction but also helpful in designing new O_2-activating metal complexes catalysts.

Oxidation of 3,5-di-*tert*-butylcatechol (DTBC, *I*) to 3,5-di-*tert*-butyl-1,2-benzoquinone (DTBQ, *II*) by O_2 is known to take place in the presence of a base. It was reported that dioximatomanganese(II) and dioximatoiron(II) complexes accelerated the triethylamine

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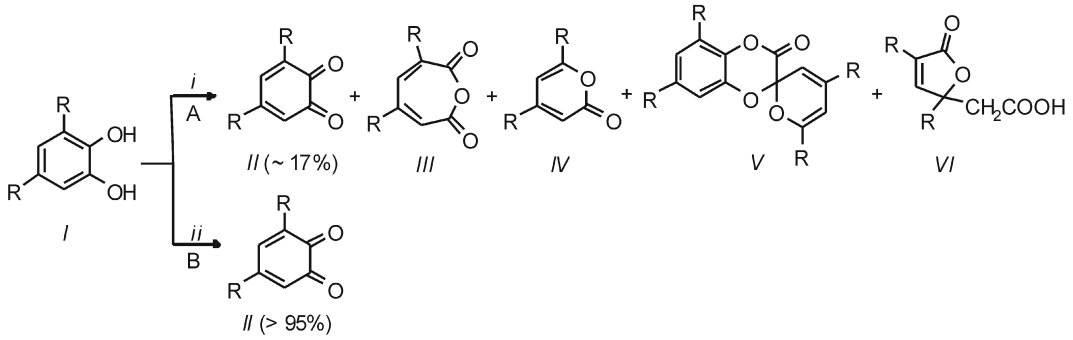

Fig. 1. Dioxygenase reaction (A) and oxidase reaction (B) of *I*. Reaction conditions: *i*) *I* (0.1 M), 1,2-dichloroethane, O_2 (0.1 MPa), $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ (10^{-5} M), 65 °C; *ii*) *I* (0.1 M), 1,2-dichloroethane, O_2 (0.1 MPa), $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ (10^{-5} M), TEA (3×10^{-4} M), 65 °C; for all compounds: R = *tert*-butyl.

(TEA)-catalysed oxidation of *I* to *II*, although these complexes alone have no or very low oxidation activity (Szigyártó et al., 2006; May et al., 2006). The general pattern for the mechanism is the formation of a ternary catalyst-O_2-substrate intermediate, which is the rate-determining step: this undergoes H-atom transfer, generating a semiquinone anionic radical. V-polyoxometalate displays dioxygenase activity in the absence of a base after an induction period. However, the V-polyoxometalate-enhanced base-catalysed oxidase reaction has not been reported. ESR is often used to determine a change in the valence state of the metal complex intermediate in order to elucidate the electron transfer pathway of the oxidation of an organic compound.

In this paper, we report that: (*i*) after an induction period, quinone, the oxidase product, instead of the dioxygenase product, is formed immediately as the major product following addition of the Lewis base into the reaction system of vanadium/*I*; (*ii*) V-polyoxometalate accelerates the base-catalysed catechol oxidase reaction rate; (*iii*) the by-product of the oxidase reaction is H_2O_2.

Experimental

Compounds *I* and *II* were obtained from Acros and Fluka, respectively. Horseradish peroxidase (POD), which was used to assess the quantity of H_2O_2, was obtained from the Huamei Biologic Engineering Co. and *N*,*N*-dialkyl-*p*-phenylenediamine (DPD) was obtained from Merck. $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ were prepared by a reported method, and the identity of the polyoxometalate was confirmed by IR spectroscopy by comparison with published data (Day et al., 1987). All other chemicals and solvents were of analytical grade and were used as received.

Separation and purification of the main products were accomplished by column chromatography, first using $CHCl_3$ as an eluent, and the acidic product was obtained by elution with methanol. The resulting five main fractions were evaporated to dryness at room temperature under vacuum and the residues were dissolved in $CDCl_3$ and then analysed by GC, ^1H and ^{13}C NMR spectroscopy. The NMR spectra were recorded on a Bruker AVANCE 400 spectrometer and compared with the published data (Weiner & Finke, 1999). GC analyses were performed on a HITACHI G-3900 GC spectrometer equipped with a FID detector and a SGE BP-5 capillary column (30 m × 0.25 mm) with the following temperature programme: initial temperature, 180 °C; heating rate, 5 °C min^{-1}; final temperature, 220 °C (final time, 20 min). The IR spectra (in KBr disc) were obtained on a TENSOR 27 FTIR spectrometer. UV-VIS spectra were recorded on a Lambda Bio 20 spectrophotometer. The H_2O_2 test was performed as follows: H_2O_2 was extracted three times from the reaction solution using deionised water and then detected by the spectrophotometric DPD method (Bader et al., 1988).

Results and discussion

When present alone in a solution, $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ catalyses neither the catechol oxidase reaction nor the catechol dioxygenase reaction immediately. However, four dioxygenation products together with ca. 17 % of *II* (the net mass balance of the five products was ∼ 95 %) were obtained after an hour's induction period and a further six hours of reaction, as shown in Fig. 1. When the Lewis base, TEA, was added to the vanadium/*I* solution, the oxidase reaction proceeded immediately and was completed after three hours with high selectivity without any discernible induction period. To clarify whether the oxidase reaction of catechol in the presence of the base occurs only in the induction period of the catechol dioxygenation reaction, namely prior to formation of the dioxygenase catalyst, TEA was added into the polyoxometalate-catalysed oxygenation reaction solution of *I* after the induction period and it was found that unreacted catechol was also autoxidised to *II* with high selectivity. The induction period in the vanadium/*I* reaction system is the time in

which the autoxidation-initiated dioxygenation catalyst is formed (Yin et al., 2005); the lack of the induction period in the presence of the base is kinetic-substantiated evidence that the vanadium complexes formed in the initial stage are the real oxidase catalyst. This suggests that the addition of the base not only restrains the formation of real dioxygenation catalyst but also destroys or deactivates the real dioxygenation catalyst formed.

On the one hand, the Lewis base quenches the V-polyoxometalate-catalysed catechol dioxygenase reaction and catalyses the oxidase reaction. On the other hand, the existence of V-polyoxometalate affects the autoxidation rate of the base-catalysed autoxidation of *I*. The autoxidation product *II* shows maximum absorption at 404 nm. The effects of V-polyoxometalate on the autoxidation of *I* are illustrated by the time-dependent UV-VIS spectra at 404 nm. It could be argued that the surface oxygen atoms of V-polyoxovanadate showed basicity and served as a bonding site for protons in some cases. However, the V-polyoxometalate could not catalyse the autoxidation of *I* in the absence of a Lewis base during the induction period. The V-polyoxometalate did not show the base-catalysis properties. The solution value of V-polyoxometalate for the first protonation constant pK_a (μ_3-O) is 5.8 (Henry, 2002) and the pK_a of *I* is 10.05 (Jovanovic et al., 1995). It would be difficult for V-polyoxometalate to acquire a proton from *I* in 1,2-dichloroethane solution. This indicates that TEA is the base catalyst for the base-catalysed autoxidation reaction. When V-polyoxometalate was added to the base-catalysed autoxidation system of *I*, remarkably, the autoxidation rate increased immediately, as shown in Fig. 2. This indicates that the V-polyoxometalate and a Lewis base can catalyse the catechol autoxidation reaction simultaneously. To the best of our knowledge, this is the first report that the V-polyoxometalate-catalysed catechol dioxygenase reaction was restrained but the oxidase reaction rate of catechol was significantly accelerated in the presence of a Lewis base.

No ESR signal was observed in the solution of the dioxygenation reaction during the induction period because no V(IV) compounds and semiquinone radicals were formed. After the induction period, a 10-line ESR spectrum centred at $g = 2.004$ was observed during the dioxygenation reaction, as shown in Fig. 3, this should be a signal of semiquinone radical complexed with vanadium. The 10-line spectrum was assigned to V(I)$_2$(II) by Morris et al. (2009). The ESR spectrum in the presence of TEA showed the characteristic signal of 3,5-di-*tert*-butyl-1,2-benzosemiquinonate anionic radical intermediate (DTBSQ) as shown in Fig. 3 (Szigyártó et al., 2006). This indicates that the semiquinone radical is not bound to the vanadium complex but exists in a free state. The oxidase reaction solution in the presence of TEA does not display

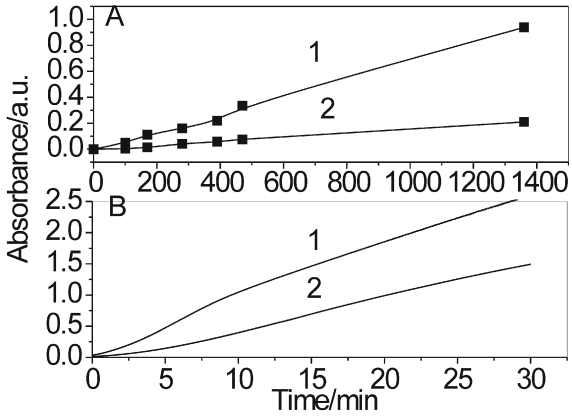

Fig. 2. Effect of V-containing polyoxometalate on the oxidation rate of *I* by monitoring the increase in absorbance at a wavelength of 404 nm of the product *II*: in the presence (1) and in the absence (2) of $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$. The added base: (A) pyridine, (B) triethylamine. $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ (10^{-5}M), *I* (0.02 M), pyridine and triethylamine (3×10^{-4} M), 25 °C.

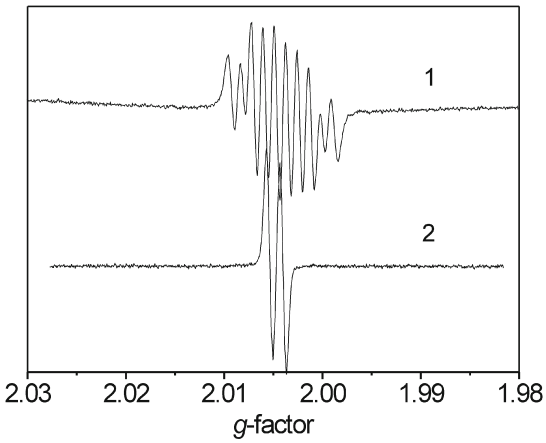

Fig. 3. ESR spectrum of dioxygenation reaction (1) and autoxidation reaction (2).

the V(IV) signal either in fluid or in frozen solution (77 K), although V(IV) showed a marked eight-line signal in the presence of TEA even at room temperature (Branca et al., 1990). The electron transfer from *I* to O_2 to form semiquinone may occur via the hydrogen bond, vanadium or via both. At present, we have no direct ESR evidence that vanadium is involved. We speculate that the hydrogen bond may play an important role in the electron transfer from *I* to O_2 in the presence of a base.

Fig. 4 shows the ESR signal changes during the oxidation of *I* upon the addition of different concentrations of TEA. When the concentration of TEA was lower than that of vanadium, the addition of TEA significantly reduced the strength of the 10-line ESR signal of the vanadium complex. However, the ESR signal

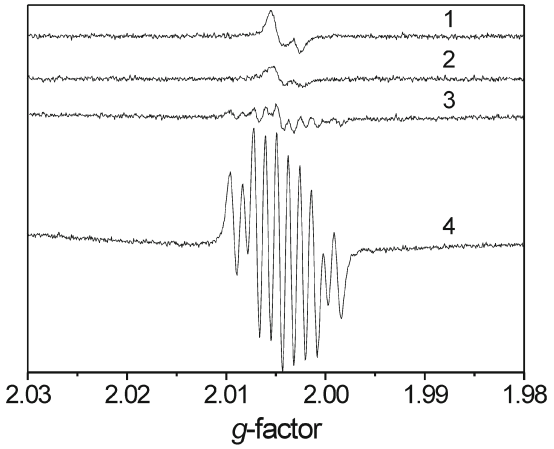

Fig. 4. ESR changes during dioxygenation after the addition of different concentrations of base, $[V_{10}O_{28}H_3]^{3-}$ (5×10^{-4} M), *I* (0.011 M); concentration of TEA: 0.014 M (1), 0.0058 M (2), 0.0014 M (3), no TEA (4).

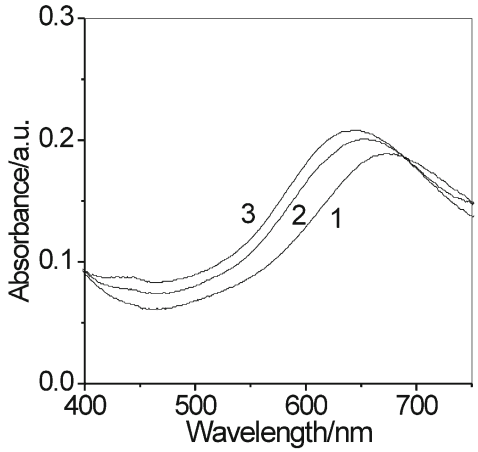

Fig. 5. Effect of oxygen on the vanadium–catechol complex spectrum: catalyst and *I*, in air (1); catalyst and *I*, deoxygen (2); catalyst, *I*, and pyridine, in air (3).

changed from the 10-line spectrum to the semiquinone radical signal after the concentration of TEA exceeded that of vanadium. This means that the addition of a sufficient amount of TEA destroys the catalyst formed in the dioxygenation reaction and restrains its regeneration through suppressing coordination of the catechol radical to the vanadium centre but remaining in a free state. In this manner, the vanadium complex and base co-catalyse the oxidase reaction of *I*.

The ESI-MS as a semi-quantitative method is capable of identifying the main species in a solution. Significantly, two common major peaks ($DTBC^-$ of m/z 221, and $V(DTBC)_3^-$ of m/z 711) are detected in the mass spectra in the presence and absence of a base. It is worth noting that the ratio of $DTBC^-/V(DTBC)_3^-$ in the presence of the base is lower than that in the absence of the base. The results show that *I* and vanadium form the $V(DTBC)_3^-$ complex first, irrespective of the presence or absence of the base, but the addition of the base facilitates the formation of a ligand anion and then facilitates the formation of the $V(DTBC)_3^-$ complex.

Under the dioxygenation reaction conditions, $V(DTBC)_3^-$ can be converted to $[VO(DTBSQ)(DTBC)]_2$ after an induction period when $[Na(CH_3OH)_2]_2[V(DTBC)_3 \cdot 4CH_3OH$ is used as the pre-catalyst (Yin & Finke, 2005). So it may be supposed that $V(DTBC)_3^-$, formed when $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ is used as the pre-catalyst, initiates the dioxygenation reaction after the induction period. No induction is observed in the oxidation reaction in the presence of the base, which means that $V(DTBC)_3^-$ and the base co-catalyse the oxidation of $DTBC^{2-}$. The experiments show that $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ first produces $V(DTBC)_3^-$, and then it performs dioxygenation after an induction period in the absence of the base, and oxidation reactions immediately in the presence of the base, respectively.

We also determined the effect of oxygen on the vanadium–catechol complex spectrum using a UV-VIS spectrometer as shown in Fig. 5. The absorption spectrum shifted towards lower energy when the complex was exposed to oxygen and shifted back to the original absorption band after deoxygenation. This indicates that oxygen forms weak bonds with the vanadium–catechol complex: the binding of oxygen causes the vanadium–catechol complex spectrum to shift and the binding of oxygen is weak enough for it to be released by deoxygenating using an inert gas. When pyridine was added to the reaction solution, oxygen had no effect on the absorption spectrum. Interestingly, the absorption spectrum of the vanadium–catechol complex in the presence of pyridine is similar to that of the vanadium–catechol complex under deoxygenation conditions. We deduced that the base occupied the binding site of oxygen in the vanadium–catechol complex so that it restrained the formation of the real dioxygenation catalyst, and further prevented the formation of dioxygenation products.

Only a small quantity ($< 2 \times 10^{-6}$ M) of H_2O_2 was detected in the V-polyoxometalate-catalysed catechol dioxygenase reaction. However, in the presence of TEA, the formation of H_2O_2 in the oxidase reaction of *I* was detected. This indicates that H_2O_2 is a by-product of the oxidase reaction. The small amount of H_2O_2 generated, concomitant with the formation of *II* product in the V-polyoxometalate-catalysed catechol dioxygenase reaction, is rapidly consumed by the vanadium catalyst (Yin et al., 2005), hence H_2O_2 was difficult to detect. However, *I* was oxidised to *II* with a high selectivity and reaction rate in the presence of TEA, which led to the formation of a larger quantity of H_2O_2. The formation rate of H_2O_2 is higher than the consumption rate of H_2O_2 in the presence of TEA, hence H_2O_2 accumulated at the initial stage of the reaction. The formation rate of H_2O_2 decreased with the

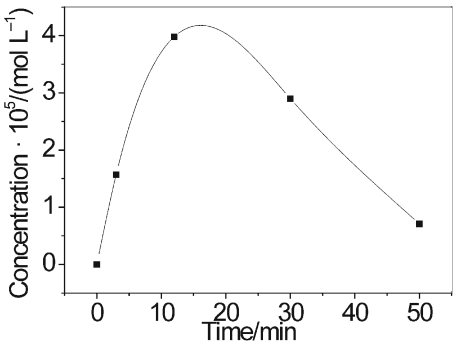

Fig. 6. H_2O_2 concentration change during oxidation reaction in the presence of TEA (3×10^{-4} M), I (0.02 M), and $[(C_4H_9)_4N]_3[V_{10}O_{28}H_3]$ (10^{-5} M).

oxidation of I but the consumption rate remained almost the same; then the H_2O_2 concentration began to decrease, as shown in Fig. 6. TEA is known to react with H_2O_2 to give Et_3NO and H_2O. However, just the catalytic amount of TEA was added during the autoxidation reaction in the research presented in this paper. TEA would have been consumed prior to the completion of the autoxidation reaction had it reacted with the H_2O_2 formed so as to give Et_3NO. Hence, we believe that the H_2O_2 decomposition was caused by V-polyoxometalate not TEA.

Conclusions

In the presence of a base, the dioxygenase reaction changes to the oxidase reaction without an induction period. ESR signals show that the semiquinone radical binds to vanadium in the dioxygenation reaction but exists in a free state in the presence of the base. V-polyoxometalate can enhance the base-catalysed catechol autoxidation reaction.

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References

Bader, H., Sturzenegger, V., & Hoigné, J. (1988). Photometric method for the determination of low concentrations of hydrogen peroxide by the peroxidase catalyzed oxidation of *N,N*-diethyl-*p*-phenylenediamine (DPD). *Water Research*, *22*, 1109–1115. DOI: 10.1016/0043-1354(88)90005-x.

Branca, M., Micera, G., Dessi, A., Sanna, D., & Raymond, K. N. (1990). Formation and structure of the tris(catecholato)vanadate(IV) complex in aqueous solution. *Inorganic Chemistry*, *29*, 1586–1589. DOI: 10.1021/ic00333a030.

Cowan, J. A. (1998). Metal activation of enzymes in nucleic acid biochemistry. *Chemical Reviews*, *98*, 1067–1088. DOI: 10.1021/cr960436q.

Cox, D. D., & Que, L., Jr. (1988). Functional models for catechol 1,2-dioxygenase. The role of the iron(III) center. *Journal of the American Chemical Society*, *110*, 8085–8092. DOI: 10.1021/ja00232a021.

Day, V. W., Klemperer, W. G., & Maltbie, D. J. (1987). Where are the protons in $H_3V_{10}O_{28}^{3-}$? *Journal of the American Chemical Society*, *109*, 2991–3002. DOI: 10.1021/ja00244a022.

Gao, X., & Xu, J. (2006). The oxygen activated by the active vanadium species for the selective oxidation of benzene to phenol. *Catalysis Letters*, *111*, 203–205. DOI: 10.1007/s10562-006-0148-1.

Groves, J. T., Bonchio, M., Carofiglio, T., & Shalyaev, K. (1996). Rapid catalytic oxygenation of hydrocarbons by ruthenium pentafluorophenylporphyrin complexes: Evidence for the involvement of a Ru(III) intermediate. *Journal of the American Chemical Society*, *118*, 8961–8962. DOI: 10.1021/ja9542092.

Hagen, C. M., Vieille-Petit, L., Laurenczy, G., Süss-Fink, G., & Finke, R. G. (2005). Supramolecular triruthenium cluster-based benzene hydrogenation catalysis: Fact or fiction? *Organometallics*, *24*, 1819–1831. DOI: 10.1021/om048976y.

Henry, M. (2002). Quantitative modelization of hydrogen-bonding in polyoxometalate chemisty. *Journal of Cluster Science*, *13*, 437–458. DOI: 10.1023/a:1020559217894.

Jovanovic, S. V., Kónya, K., & Scaiano, J. C. (1995). Redox reactions of 3,5-di-*tert*-butyl-1,2-benzoquinone. Implications for reversal of paper yellowing. *Canadian Journal of Chemistry*, *73*, 1803–1810. DOI: 10.1139/v95-222.

Koval, I. A., Gamez, P., Belle, C., Selmeczi, K., & Reedijk, J. (2006). Synthetic models of the active site of catechol oxidase: mechanistic studies. *Chemical Society Reviews*, *35*, 814–840. DOI: 10.1039/b516250p.

Lin, G., Reid, G., & Bugg, T. D. H. (2001). Extradiol oxidative cleavage of catechols by ferrous and ferric complexes of 1,4,7-triazacyclononane: Insight into the mechanism of the extradiol catechol dioxygenases. *Journal of the American Chemical Society*, *123*, 5030–5039. DOI: 10.1021/ja004280u.

May, Z., Simándi, L. I., & Németh, Z. (2006). A novel iron-enhanced pathway for base-catalyzed catechol oxidation by dioxygen. *Reaction Kinetics and Catalysis Letters*, *89*, 349–358. DOI: 10.1007/s11144-006-0147-7.

Morris, A. M., Pierpont, C. G., & Finke, R. G. (2009). Dioxygenase catalysis by d^0 metal–catacholate complexes containing vanadium and molybdenum with H_2(3,5-DTBC) and H_2(3,6-DTBC) substrates. *Journal of Molecular Catalysis A: Chemical*, *309*, 137–145. DOI: 10.1016/j.molcata.2009.05.008.

Ragsdale, S. W., & Kumar, M. (1996). Nickel-containing carbon monoxide dehydrogenase/acetyl-CoA synthase. *Chemical Reviews*, *96*, 2515–2540. DOI: 10.1021/cr950058+.

Sigel, R. K. O., & Pyle, A. M. (2007). Alternative roles for metal ions in enzyme catalysis and the implications for ribozyme chemistry. *Chemical Reviews*, *107*, 97–113. DOI: 10.1021/cr0502605.

Szigyártó, I. C., Simándi, L. I., Párkányi, L., Korecz, L., & Schlosser, G. (2006). Biomimetic oxidation of 3,5-di-*tert*-butylcatechol by dioxygen via Mn-enhanced base catalysis. *Inorganic Chemistry*, *45*, 7480–7487. DOI: 10.1021/ic060618v.

Weiner, H., & Finke, R. G. (1999). An all-inorganic, polyoxometalate-based catechol dioxygenase that exhibits >100 000 catalytic turnovers. *Journal of the American Chemical Society*, *121*, 9831–9842. DOI: 10.1021/ja991503b.

Yin, C. X., & Finke, R. G. (2005). Vanadium-based, extended catalytic lifetime catechol dioxygenases: Evidence for a common catalyst. *Journal of the American Chemical Society*, *127*, 9003–9013. DOI: 10.1021/ja051594e.

Yin, C. X., Sasaki, Y., & Finke, R. G. (2005). Autoxidation-product-initiated dioxygenases: vanadium-based, record catalytic lifetime catechol dioxygenase catalysis. *Inorganic Chemistry*, *44*, 8521–8530. DOI: 10.1021/ic050717t.