ACS Catalysis

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# Quantitative 1,10-Phenanthroline Catalyst-Poisoning Kinetic Studies of Rh(0) Nanoparticle and $Rh_4$ Cluster Benzene Hydrogenation Catalysts: Estimates of the Poison $K_{association}$ Binding Constants, of the Equivalents of Poison Bound and of the Number of Catalytically Active Sites for Each Catalyst

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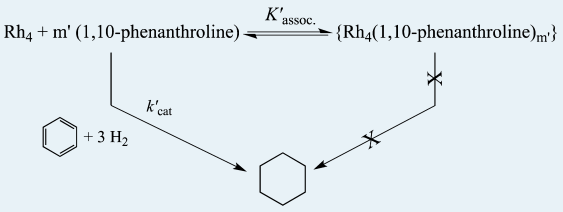
Supporting Information

**ABSTRACT:** Quantitative catalyst poisoning studies are of fundamental interest and importance because (a) knowledge of the number of true active sites is required for calculation of the *true* turnover frequency = (moles of product)/(moles of actual *active sites*)(time), and because (b) quantitative catalyst poisoning is proving to be a key, required piece of data en route to distinguishing single metal ($M_1$), small metal cluster (e.g., $M_4$), or metal nanoparticle ($M_n$) catalysis. In evidence of the latter point, quantitative catalyst poisoning experiments using 1,10-phenanthroline as the poison proved to be crucial in the recent identification of $Rh_4$ subnanometer clusters as the true benzene hydrogenation catalyst in a system beginning with $[RhCp^*Cl_2]_2$ ($Cp^*$: ($\eta^5$-$C_5(CH_3)_5$)) at 100 °C and 50 atm initial $H_2$ pressure (Bayram et al. *J. Am. Chem. Soc.* **2011**, *133*, 18889). However and despite the success of those quantitative poisoning studies, five questions about such poisoning studies remained unanswered, questions posed and then addressed herein. In addition, the analysis herein of the 1,10-phenanthroline poisoning of both Rh(0) nanoparticle and $Rh_4$ subnanometer benzene hydrogenation catalysts results in kinetic models for, respectively, strong-binding and weak-binding poisons. Also provided are quantitiative estimates of the poison binding constants, of the number of equivalents required to completely poison each catalyst, and of the number of active sites on each catalyst. The weak-binding poison kinetic model is then shown to have immediate applicability toward analyzing extant literature data via its application to literature $CS_2$ quantitative poisoning data for ammonia-borane dehydrocoupling beginning with a [Ru(cod)(cot)] (cod: cyclooctadiene and cot: cyclooctatriene) precatalyst. The significance of the results is then summarized in a Conclusions section.

Minimalistic Kinetic Model for Weak-Binding Poisons Plus $Rh_4$ Sub-Nanometer Clusters




**KEYWORDS:** *catalyst, poisoning studies, kinetics and mechanism, 1,10-phenanthroline catalyst poisoning, rhodium catalysis, nanoparticles, subnanometer clusters, benzene hydrogenation, determination of the true catalyst*

## INTRODUCTION

Catalyst poisoning is a fundamental and important topic to any and all catalysis.[1−9] Indeed, one cannot even calculate a true turnover frequency, defined as TOF = moles of product/(moles of catalytically active sites−time), without knowledge of the true number of catalytically active sites. Moreover, quantitative catalyst poisoning experiments are proving increasingly important in the identification of the true catalyst in a given reaction, a task that can involve distinguishing single-metal homogeneous from smaller metal cluster and larger, polymetallic nanoparticle catalysis.[10,11] Poisoning studies are proving powerful in distinguishing such classes of catalysts since on going from a single metal, single-active-site homogeneous catalyst to a heterogeneous, nanoparticle catalyst, the required equiv of poison per total equiv of metal present needed to deactivate completely the catalyst typically decreases from ≥1 to ≪1.[10,11]

Recently, quantitative catalyst poisoning studies using 1,10-phenanthroline as the poison proved crucial in identifying subnanometer $Rh_4$ clusters of average composition $Rh_4Cp^*_{2.4}Cl_4H_c$ (hereafter abbreviated as $Rh_4$) as the true catalysts in benzene hydrogenation performed at 100 °C and 50 atm initial $H_2$ pressure beginning from $[RhCp^*Cl_2]_2$ as the precatalyst.[12] In that study, in operando[13] X-ray absorption fine structure (XAFS) studies showed that 98 ± 2% of the initial Rh present in the $[RhCp^*Cl_2]_2$ precatalyst evolved to $Rh_4$ clusters; however, the 70-fold faster reactivity of model polyethyleneglycol-dodecylether hydrosol stabilized Rh(0) nanoparticles studied in control reactions meant that Rh(0) nanoparticles would have been the dominant and kinetically competent catalysts if even about ≥1.4% of the initial $[RhCp^*Cl_2]_2$

**Received:** May 28, 2012
**Revised:** July 30, 2012
**Published:** August 17, 2012

precatalyst had been converted to the model (or similar activity) Rh(0) nanoparticles. Significantly, 1,10-phenanthroline quantitative catalyst poisoning experiments, reproduced in Figures 1 and 2 herein, were, in the end analysis, what

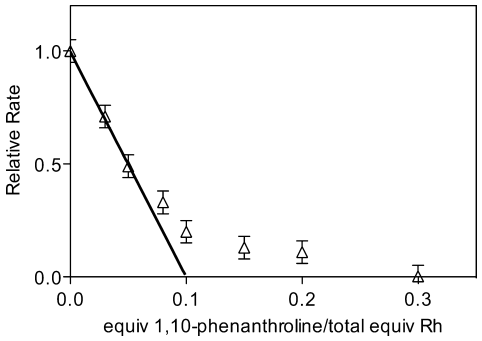



**Figure 1.** Plot of the relative rate vs equiv of 1,10-phenanthroline per equiv of total rhodium present for benzene hydrogenation beginning with model polyethyleneglycol-dodecylether hydrosol stabilized Rh(0) nanoparticles at 100 °C and 50 atm initial $H_2$ pressure. The value of the $x_{intercept}$ is 0.10 ± 0.02 for the straight line drawn.[14]

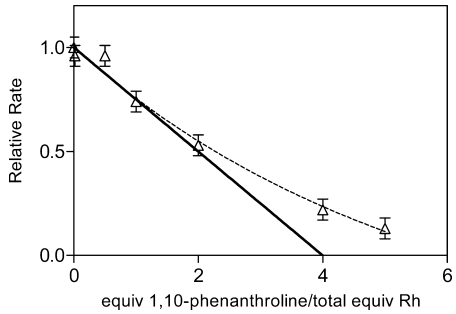



**Figure 2.** Plot of the relative rate vs equiv of 1,10-phenanthroline per equiv of total, fully evolved rhodium present in the form of 98 ± 2% $Rh_4$ clusters identified via in operando-XAFS[12] for benzene hydrogenation at 100 °C and 50 atm initial $H_2$ pressure. The value of the $x_{intercept}$ is 4.0 ± 0.4 for the tangent, straight line drawn. An important question addressed in this paper is whether this prior-literature recommended way of analyzing this data, by a linear extrapolation method to yield a resultant intercept, is correct or should be replaced by the weak-binding poison model presented herein.

distinguished $Rh_4$ clusters as the true catalyst from larger, 2−3 nm Rh(0) nanoparticles as an alternative hypothesis for the true catalyst.[12]

The poisoning data in Figures 1 and 2 were analyzed previously[12] via the common, literature-recommended practice[1,11] of drawing straight lines to the linear portion of the plot to find $x_{intercept}$. The $x_{intercept}$ for the Rh(0) nanoparticles[14] is 0.10 ± 0.02, Figure 1, whereas, for the $Rh_4$ clusters $x_{intercept}$ is 4.0 ± 0.4, Figure 2—the $Rh_4$ subnanometer cluster catalyst requiring significantly more poison in comparison to the Rh(0) nanoparticle catalyst, as expected since only a fraction of the total Rh in the nanoparticle case is on the surface, and thus accessible. Hence and as already mentioned, the quantitative 1,10-phenanthroline poisoning experiments proved to be crucial in identifying $Rh_4$ subnanometer clusters as the true benzene hydrogenation catalyst when beginning with the $[RhCp^*Cl_2]_2$ precatalyst.[12]

Despite the valuable, seemingly definitive nature[11,15] of the quantitative poisoning studies in Figures 1 and 2, five questions remain to be addressed following the prior work.[12] First, (i) what does the $x_{intercept}$ value of 0.10 in Figure 1 really mean in terms of the amount of poison required to deactivate the nanoparticles completely? Is a more rigorous, quantitative interpretation of such poisoning curves possible? Second, (ii) although the approximately linear dependence of Rh(0) nanoparticle activity on the equiv of 1,10-phenanthroline implies a strong association between Rh(0) nanoparticles and 1,10-phenanthroline, Figure 1, can one estimate a quantitative value for the binding constant of the 1,10-phenanthroline poison to the Rh(0) nanoparticle catalyst, $K_{association}$ (hereafter abbreviated as $K_{assoc.}$)?[16,17] Also, can the $K'_{assoc.}$ for 1,10-phenanthroline binding to the $Rh_4$ clusters also be obtained? Third, (iii) can one estimate a narrower range of values for the ratio of poison to the number of catalytic sites deactivated than, for example, the previously reported[11] $CS_2$/Rh(0) ratios of about 1/1.5 to 1/20? This ratio is needed to calculate the number of active sites, and thus the true turnover frequency, from the experimentally observed ratio of $CS_2$:Rh(0) observed to fully poison the catalyst. We previously identified this ratio as the "Achilles Heel" of otherwise powerful catalyst poisoning studies aimed at determining the true number of catalytically active sites.[11] Fourth and significantly, (iv) since a closer look at the poisoning curve in Figure 2 *shows a slightly sigmoidal shape*, qualitatively implying a smaller $K_{assoc.}$ constant compared to Figure 1, is the use of this classic "straight-line extrapolation"[1,11] method, and resultant $x_{intercept}$, not justified in that case as it seems? Can a more appropriate, quantitative kinetic poisoning model be applied, and if so, what does constructing such a weak-binding poison kinetic model teach us? Fifth and finally, (v) what is the best method(s) of analyzing nanoparticle and subnanometer cluster catalysts poisoning data obtained in solution? Solid−gas phase, supported nanoparticle catalyst poisoning data are traditionally and commonly handled by Langmuir adsorption isotherms,[16−20] while enzyme poisoning data are analyzed by a Michaelis−Menten kinetic treatments.[21−26]

To start, a careful search of the literature relevant to the five questions above yielded the following literature insights as a foundation from which to build the present contribution. First, historically,[1] quantitative poisoning data (plotted typically as catalyst activity vs concentration of the poison, with tangential straight lines being drawn; see Figure 8 elsewhere[1]) were then treated by Maxted using the linear equation $k_c = k_0(1 - \propto c)$, where $k_0$ is the activity without any poison present, $k_c$ is the activity when $c$ concentration of poison is present, and $\propto$ is the relative susceptibility of different catalysts to a poison under, ideally, otherwise identical conditions.[1] Later, others[27,28] and we[11] analyzed quantitative catalyst poisoning data via plots of the relative rate vs equiv of poison per total equiv of metal present, with an eye here toward making apparent the number of equivalents of poison required to poison the total metal present. Again tangential straight lines were drawn which were now analyzed by the simple, classic expression $y = -ax+b$ where $y$ is the relative rate, $-a$ is the slope of the resultant line, $x$ is the equiv of poison per total equiv of metal present, and $b$ is 1, as in Figures 1 and 2, vide supra, a treatment that is equivalent to Maxted's equation[1] if $y$ = relative rate = $k_c/k_0$ and $-\propto c = -ax$. The value of this treatment of the data is that the $x_{intercept}$ (i.e., value of $x$ when $y = 0$) provides an estimate of the equiv of poison per total metal present required to fully poison the catalyst. However, the $x_{intercept}$ is only an estimate in the more general case where the poisoning plot is not strictly linear—that is, when one is drawing a straight-line tangent to


dx.doi.org/10.1021/cs300330c | ACS Catal. 2012, 2, 1967−1975

the curved plot, as in Figures 1 and 2. A more rigorous, quantitative interpretation of $x_{intercept}$ in this more general case is lacking, but is addressed herein.

Returning to what else can be gleaned from the catalyst poisoning literature, catalyst poisoning data have been treated extensively in the chemical engineering literature,[5] including the extraction of thermodynamic data (such as $K_{assoc.}$) via engineering models focused on industrial catalysts and their reactors. However, those typically reactor-based studies (a) necessarily include variables such as (but not limited to) reactor type, flow gas rate, and catalyst bed type, and often for solid−gas phase catalytic reaction conditions;[29−31] (b) usually are, therefore, specific to a given system with resultant complex mathematical equations that obfuscate ready interpretation of the underlying, basic chemistry;[4,5,32] and hence and as Bartholomew has noted[33] (c) provide "comprehensive mathematical models that will enable more effective design and optimization of the processes deactivating catalysts",[33] but do not provide understanding of the extant poisoning phenomena at the molecular level[34−38]—the latter being the goal of quantitative catalyst poisoning *mechanistic* studies such as the present work.

Unfortunately but not surprisingly, phenomenology based words and nomenclature have arisen in the chemical engineering literature from such nonmechanistic treatments, for example the term of "antiselective poisoning"[5] (really just sigmoidal poisoning curves signifying relatively weak poisoning binding), just to pick one example, nomenclature that further obfuscates what is really occurring chemically. This is not a trivial point. The use of phenomenological, "physical" models, in place of disproof-based mechanistic models, in science is an insidious problem that often results in the wrong concepts and words being used,[39] in the final analysis, to (incorrectly) describe the resultant chemistry. More on this important topic of model building in science is available elsewhere[39] for the interested reader.

Herein, we address the questions (i)−(v) raised above in-so-far as possible by (a) deriving and justifying the $x_{intercept}$ term rigorously en route to calculating the required amount of poison (i.e., variable $m$ in what follows) needed to deactivate the catalyst completely, (b) estimating the average $K_{assoc.}$, and (c) estimating the number of catalytically active surface sites, with the first part of what follows focusing on the 1,10-phenanthroline quantitative poisoning data[12] for Rh(0) nanoparticle catalyzed benzene hydrogenation at 100 °C and 50 atm initial $H_2$ pressure. We also (d) propose a mechanism-based kinetic model from which to analyze rigorously the 1,10-phenanthroline quantitative poisoning data for $Rh_4$ cluster-based benzene hydrogenation at 100 °C and 50 atm initial $H_2$ pressure, an example of the probably more general case where a slightly sigmoidal poisoning plot is obtained, cases where drawing straight-line tangents makes little sense. The resultant kinetic model and quantitiative analysis of the poisoning data then (e) allows us to extract the required amount of poison to deactivate $Rh_4$ cluster catalyst completely (i.e., $m'$), and (f) estimates of the quantitative $K'_{assoc.}$. Finally, (g) literature $CS_2$ quantitative poisoning data for ammonia-borane dehydrocoupling beginning with a [Ru(cod)(cot)] precatalyst are analyzed using the weak-binding poison kinetic model developed herein, results which demonstrate the immediate applicability of that poisoning kinetic model.

## EXPERIMENTAL SECTION

**Materials.** Benzene (Aldrich, 99.8%, anhydrous, packaged under $N_2$), 2-propanol (Aldrich, 99.5%, anhydrous, packaged under $N_2$), and 1,10-phenanthroline (Aldrich, 99%) were transferred to and stored in a drybox, then used as received. Hydrogen gas (General Air, 99.5%) was used as received. Rh(0) nanoparticles (polyethyleneglycol-dodecylether hydrosol stabilized, ∼9 wt %-Rh, ∼2 nm Rh(0) nanoparticles) were purchased from Strem Chemicals, stored in the drybox, and used as received.

**General Procedures for Quantitative 1,10-Phenanthroline Poisoning Experiments with Rh(0) Nanoparticles and $Rh_4$ Clusters.** All experimental preparations and manipulations were performed under oxygen- and moisture-free conditions in a Vacuum Atmosphere $N_2$-drybox (<2 ppm of $O_2$ as continuously monitored by a Vacuum Atmosphere $O_2$-monitor). All quantitative 1,10-phenanthroline poisoning experiments for benzene hydrogenation reaction with either Rh(0) nanoparticles or $Rh_4$ clusters were performed in a Parr pressure reactor (model 4561) made of Monel 400 alloy. The reactor is equipped with a pressure gauge marked at intervals of 20 psig (∼1.36 atm) and an automatic temperature controller (±3 °C). The inside of the reactor contains a stainless steel (i.e., non-Monel) impeller, thermocouple, cooling loop, and dip tube, all of which are in contact with the reaction solution. A glass-liner was used to avoid contacting the reaction solution with the rest of the reactor. The glass-liner was dried overnight in a 160 °C drying oven before being transferred into the drybox and prior to use. Pressurizing the reactor took about 1 min, and $t$ = 0 was set after this time and once the reactor was fully pressurized. Pressure gauge readings vs time data were then collected and recorded manually.

**1,10-Phenanthroline Quantitative Poisoning Experiments in Benzene Hydrogenation Beginning with Polyethyleneglycol-dodecylether Hydrosol Stabilized Rh(0) Nanoparticles.** Recently reported[12] relative rate data for the quantitative poisoning of polyethyleneglycol-dodecylether hydrosol stabilized Rh(0) nanoparticles were used with three additional experiments being added. For those additional experiments, the same experimental procedure was repeated as detailed elsewhere[12] (in the Experimental Section titled "1,10-Phenanthroline Quantitative Poisoning Experiments for Polyethyleneglycol-dodecylether Hydrosol Stabilized Rh(0) Nanoparticles"[12]), but now using the addition of 0.03, 0.08, and 0.15 equiv of 1,10-phenanthroline per total rhodium (1.1, 2.9, and 5.4 mg of 1,10-phenanthroline, respectively) to the initial solution in three separate, additional poisoning experiments. The resultant hydrogenation curves for each trial were fit to a polynomial, and the initial rate was calculated as detailed previously[12] (in the Experimental Section titled "Kinetic Data Treatment: Initial Rate Method") and as shown here in the Supporting Information, Figure SI-3. Each poisoning trial was repeated three times and yielded identical initial rates within ±15% experimental error. The other 1,10-phenanthroline poisoning hydrogenation curves and initial rates are available elsewhere in ref 12 as Supporting Information, Figure SI-9.

As a control to see if the 1,10-phenanthroline poison could be hydrogenated at all by the Rh(0) nanoparticles (i.e., at longer times and if used as the hydrogenation substrate), the following experiment was done. A separate, 1,10-phenanthroline quantitative poisoning experiment as detailed above was performed using the addition of 36 mg of 1,10-phenanthroline


dx.doi.org/10.1021/cs300330c | ACS Catal. 2012, 2, 1967−1975

(1.0 equiv of 1,10-phenanthroline per total rhodium) with one change: no benzene was added; that is, 1,10-phenanthroline is the potential hydrogenation substrate in this control experiment. No hydrogen uptake was observed over 7 h, even though it it takes <15 min to completely hydrogenate 4 mL of benzene under Standard Conditions (ca. 222 equiv of benzene per total Rh; see Figure 9 elsewhere[12]). This control experiment shows that it is unlikely that much if any 1,10-phenanthroline (≤1% in this particular experiment) gets hydrogenated under the experimental condition, as expected since it is a poison.

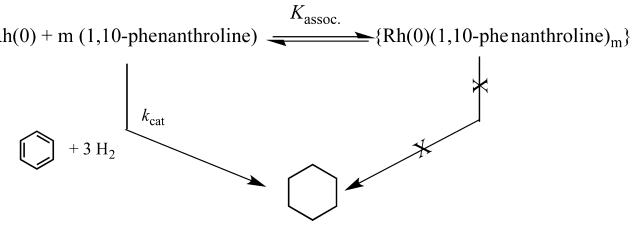
**1,10-Phenanthroline Quantitative Poisoning Experiments for Benzene Hydrogenation Beginning with, on Average, $Rh_4Cp^*_{2.4}Cl_4H_c$ Clusters.** Recently reported[12] relative rate data for the quantitative poisoning of $Rh_4Cp^*_{2.4}Cl_4H_c$ clusters with 1,10-phenanthroline were used. See Figure 7, Figures SI-8(b-f), and the experimental procedures reported in ref 12.

**Data Handling.** The nonlinear least-squares fit of the experimental data for the 1,10-phenanthroline quantitative kinetic poisoning of the $Rh_4$ clusters was performed using GraphPad Prism version 5 for Mac OS X, GraphPad Software, San Diego California U.S.A., www.graphpad.com.

## RESULTS AND DISCUSSION

**Analysis of 1,10-Phenanthroline Poisoning of Rh(0) Nanoparticles.** *Correlation of $x_{intercept}$ with the Amount of Poison "m" Required to Deactivate the Catalyst Completely.* The quantitative poisoning plot of polyethyleneglycol-dodecylether hydrosol stabilized Rh(0) nanoparticles with 1,10-phenathroline is given in Figure 1. The relative rate initially decreases linearly with increasing number of poison equiv, as is commonly seen in the literature.[1,11] The typical literature practice[1,11] of the linear regression analysis of this linear portion of the plot yields $x_{intercept}$ of 0.10 ± 0.02, Figure 1.[14] To justify the $x_{intercept}$ as well as analyze the basic underlying chemistry, a minimalistic 1,10-phenanthroline poisoning scheme is proposed, Scheme 1. A full derivation of the kinetics corresponding to Scheme 1, eq 1, is provided in the Supporting Information.

**Scheme 1. Minimalistic, Strong-Binding Poison Kinetic Model for 1,10-Phenanthroline Poisoning of Rh(0) Nanoparticles**




$$\text{relative rate} = \left\{-\frac{1}{m}\right\}\left\{\frac{[\text{1,10-phenanthroline}]_{\text{initial}}}{[\text{Rh(0)}]_{\text{initial}}}\right\} + 1 \quad (1)$$

Note that in this minimalistic model for a strong-binding poison (as well as in the also minimal model for a weak-binding poison in Scheme 2, vide infra), issues such as the individual binding constants of the net $m$ equiv of poison, or the structures of those bound poisons, are beyond the scope of this work—the goal of this paper being to provide clear, minimal, mechanistic schemes for the quantitative fitting, and initial interpretation, of metal $M(0)_n$ nanoparticle and $M_4$ or related cluster, poisoning data.

Briefly, the initially linear decrease in catalytic activity with added 1,10-phenanthroline implies a strong association between the Rh(0) nanoparticles and the 1,10-phenanthroline, one where all the added 1,10-phenanthroline binds to the Rh(0) nanoparticles, at least in the initial, linear region. Hence, in the initial linear region, the initial 1,10-phenanthroline concentration will be equal to the poisoned catalyst concentration, that is, $[\text{1,10-phenanthroline}]_{\text{initial}} \approx m[\{\text{Rh(0)-(1,10-phenanthroline)}_m\}]$. The resultant relative rate equation is then eq 1, which is in the form of the standard linear function: $y = ax + b$, where $y$ is the relative rate; $a$ is the slope, $(-1/m)$; $x$ is $\{[\text{1,10-phenanthroline}]_{\text{initial}}/[\text{Rh(0)}]_{\text{initial}}\}$, namely, the equiv of poison per equiv of total metal present;[40] and $b$ is 1.

The linear regression analysis of the initially linear portion of Figure 1 yields $y = -9.9x + 1$ with an $x_{\text{intercept}}$ equal to 0.10. The slope of the line is $-(1/m) = -9.9$ making $m = 0.10$, $m$ being the required equiv of 1,10-phenanthroline per total equiv of metal needed to deactivate the catalyst completely. Hence, $m$ is equal to $x_{\text{intercept}}$.

Overall, the analysis of Scheme 1 reveals the $x_{\text{intercept}}$ $(= m)$ is indeed the amount of 1,10-phenanthroline required to deactivate completely the Rh(0) nanoparticle catalyst on a per Rh(0) present basis, as expected given the tight binding of the poison, $[\text{1,10-phenanthroline}]_{\text{initial}} \approx m[\{\text{Rh(0)(1,10-phenanthroline)}_m\}]$ assumption used in the derivation of eq 1. There is, then and also, an equivalence between eq 1 and the $k_c = k_0(1 - \propto c)$ equation used classically to treat strong-binding poisoning data:[1] the two are equivalent if the relative rate equals $k_c/k_0$ so that, therefore, also $\propto c = (1/m)\{[\text{1,10-phenanthroline}]_{\text{initial}}/[\text{Rh(0)}]_{\text{initial}}\}$.

*Estimate of the Approximate $K_{assoc.}$* One can also in principle calculate the $K_{\text{assoc.}}$ in Scheme 1 via eq 2 where now, for simplification in writing the equilibrium expression, the amount of poisoned catalyst, $[\{\text{Rh(0)(1,10-phenanthroline)}_m\}] = [\text{P}]$, $[\text{Rh(0)}]_{\text{initial}} = [\text{A}_0]$, and $[\text{1,10-phenanthroline}]_{\text{initial}} = [\text{B}_0]$. However, recalling the $[\text{1,10-phenanthroline}]_{\text{initial}} \approx m[\{\text{Rh(0)(1,10-phenanthroline)}_m\}]$ (or, now, equivalently in the simplified nomenclature $[\text{B}_0] \approx m[\text{P}]$) assumption used to derive eq 1, vide supra, the $[\text{B}_0] - m[\text{P}]$ term in the denominator of eq 2 is approximately zero, and as a result $K_{\text{assoc.}}$ "blows up" and becomes undefined.

$$K_{\text{assoc.}} = \frac{[\text{P}]}{\{[\text{A}_0] - [\text{P}]\}\{[\text{B}_0] - m[\text{P}]\}^m} \quad (2)$$

However, one can calculate via eq 3 the poisoned catalyst concentration, [P], from the experimentally determined relative rate values. Then, substituting the eq 3 [P] values into eq 2 followed by simplification yields eq 4 (see the Supporting Information for details). Then, using the experimentally determined $m = 0.1$ value, an estimate of $K_{\text{assoc.}} \leq 1.4\ \text{M}^{-0.10}$ is obtained via eq 4. Note that the unusual units $(\text{M}^{-0.10})$ of $K_{\text{assoc.}}$ in this strong-binding case mean that this specific $K_{\text{assoc.}}$ can be compared quantitatively only to other $K_{\text{assoc.}}$ that have identical $m$ values (i.e., and thus identical units). Worth noting here is that, as one might expect, 1,10-phenanthroline is known to bind relatively tightly to other metal nanoparticles. For example, 1,10-phenanthroline binds tightly to Pd nanoparticles


dx.doi.org/10.1021/cs300330c | ACS Catal. 2012, 2, 1967−1975

with polyvinyl pyrrolidone (PVP) as an added stabilizer employed in olefin hydrogenations.[41]

$$[P] = [A_0] \times (1 - \text{relative rate}) \quad (3)$$

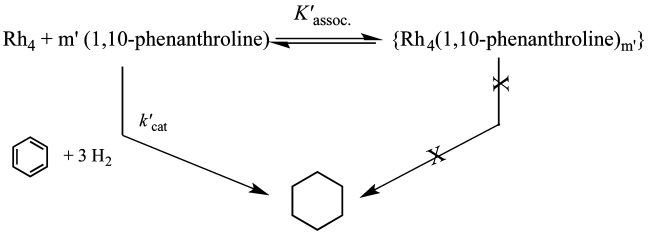
$$K_{\text{assoc.}} = \frac{\{1 - \text{relative rate}\}}{\{\text{relative rate}\}\{[B_0] - m\{[A_0]\{1 - \text{relative rate}\}\}\}^m} \quad (4)$$

*Estimate of the Number of the Catalytically Active Sites.* The Rh(0) nanoparticle diameters are 2−3 nm according to TEM analysis (see Supporting Information, Figure SI-6, elsewhere in ref 12), which in turn corresponds to $Rh(0)_{\sim 300}$ and $Rh(0)_{\sim 1100}$ nanoparticles, respectively,[42,43] that is, on average $Rh(0)_{\sim 700}$ nanoparticles for the purposes of the following estimate of the number of catalytically active sites. Such average $Rh(0)_{700}$ nanoparticles have a bit more than ∼40%, about 300 of their total rhodium present on the surface,[42,43] where catalysis occurs. With the assumption that one 1,10-phenanthroline poisons one surface rhodium (i.e., if one assumes a Rh:1,10-phenanthroline ratio is 1:1), then the $x_{\text{intercept}}$ of 0.10 equiv of 1,10-phenanthroline poison per total rhodium becomes 0.25 equiv of 1,10-phenanthroline per total *surface* rhodium. (If, on the other hand, two or three catalytically active sites are poisoned by one 1,10-phenanthroline simultaneously, then, the calculated fraction of catalytically active surface Rh atoms becomes 0.50 and 0.75, respectively.) Noteworthy here is that simultaneous deactivation of four surface rhodium atoms by one 1,10-phenanthroline is an upper limit to the possible poisoning since, the observed surface Rh:1,10-phenanthroline ratio is 4:1 The possibility of 100% of the surface Rh being active is improbable, however, since that would require completely unligated, completely "naked nanoparticles",[44] which are unknown.[44] Hence, the useful implication is that one 1,10-phenanthroline molecule poisons between 1 and 3, and rigorously ≤4, surface Rh atoms, at least under these specific conditions of our benzene hydrogenation experiments and within the $Rh(0)_{\sim 700}$ average nanoparticle size assumption.

**Quantitative Analysis of 1,10-Phenanthroline Poisoning of $Rh_4$ Clusters.** The poisoning plot of $Rh_4$ clusters by 1,10-phenanthroline, Figure 2, is slightly but detectably sigmoidal, implying a weaker association of 1,10-phenanthroline to the $Rh_4$ clusters and concomitant smaller $K'_{\text{assoc.}}$, Scheme 2. Quantitatively, when $[Rh_4]_{\text{initial}} = 1.15 \times 10^{-3}$ M and $[1,10\text{-phenanthroline}]_{\text{initial}} = 2.3 \times 10^{-3}$ M, a relative rate of 0.96 was observed[12]—that is, the benzene hydrogenation catalytic activity is virtually unaffected, yielding the same relative rate within ±10% experimental error to that seen *without* any 1,10-phenanthroline addition. This is arguably consistent with homogeneous catalysts by the $Rh_4Cp^*_{2.4}Cl_4H_o$,[12] such homogeneous catalysts having been previously claimed to be less sensitive to poisons,[45,46] at least when sterically bulky ligands[1] such as the Cp* in $Rh_4Cp^*_{2.4}Cl_4H_c$ are present. The most important initial point here, then, is that the classic, linear treatment of fitting with a straight line (i.e., with $k_c = k_0(1 - \propto c)$ or its relative rate vs equiv of poison per equiv of metal catalyst version, eq 1) is inappropriate and should not be used[1,11] because of the nonlinear, sigmoidal nature of the poisoning plot, Figure 2.

Scheme 2 presents an alternative, minimalistic kinetic model from which to analyze the 1,10-phenanthroline poisoning of the $Rh_4$ clusters under the weak-binding assumption where also experimentally $[1,10\text{-phenanthroline}]_{\text{initial}} \gg m'[\{Rh_4(1,10\text{-phenanthroline})_{m'}\}]$ so that $[1,10\text{-phenanthroline}]_{\text{initial}} \approx [1,10\text{-phenanthroline}]_{\text{equilibrium}}$. Equation 5 gives the resultant relative rate expression, with now its $K'_{\text{assoc.}}$ and $m'$ constants that were used to analyze quantitatively the poisoning data in Scheme 2 for 1,10-phenanthroline poisoning of the $Rh_4$

**Scheme 2. Minimalistic, Weak-Binding Poison Kinetic Model for 1,10-Phenanthroline Poisoning of $Rh_4$ Clusters of Average Stoichiometry $Rh_4Cp^*_{2.4}Cl_4H_c$[12]**




clusters. The Supporting Information provides the full details of the derivation of eq 5.

$$\text{relative rate} = \frac{1}{1 + K'_{\text{assoc.}}[1,10\text{-phenanthroline}]^{m'}_{\text{initial}}} \quad (5)$$

A nonlinear least-squares fit using eq 5 of the experimental poisoning data back in Figure 2 *yields a good fit to the data*, $R^2 = 0.993$, Figure 3.

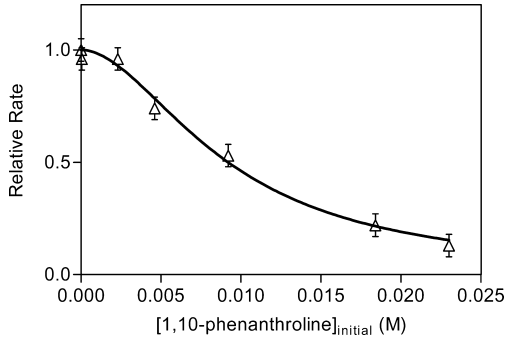



**Figure 3.** The relative rate vs 1,10-phenanthroline concentration data for $Rh_4$ clusters (Δ) and fit to the data using eq 5 (solid line), $R^2 = 0.993$, with resultant $K'_{\text{assoc.}} = 6.1 \pm 4.4 \times 10^3$ $M^{-1.86}$ and $m' = 1.86 \pm 0.15$. The data in this figure are the identical data as in Figure 1, but now the *x*-axis is the $[1,10\text{-phenanthroline}]_{\text{initial}}$ concentration required for the curve-fitting by eq 5 (i.e., and not the equiv 1,10-phenanthroline per total equiv Rh present, Figure 1).

The good fit seen in Figure 3 supports the assumption of $[1,10\text{-phenanthroline}]_{\text{initial}} \approx [1,10\text{-phenanthroline}]_{\text{equilibrium}}$ that was used to derive eq 5 and as detailed in the Supporting Information. In addition, ex-post-facto calculations support the $[1,10\text{-phenanthroline}]_{\text{initial}} \approx [1,10\text{-phenanthroline}]_{\text{equilibrium}}$ assumption by showing that ≥87% of $[1,10\text{-phenanthroline}]_{\text{initial}}$ is $[1,10\text{-phenanthroline}]_{\text{equilibrium}}$ for each value of $[1,10\text{-phenanthroline}]_{\text{initial}}$ actually used (the Supporting Information presents the details of these calculations for the interested reader).

*Estimates of the $K_{assoc}$. Value and the Number of the Catalytically Active Sites.* As noted above, the association constant for the 1,10-phenanthroline binding to the $Rh_4$


dx.doi.org/10.1021/cs300330c | ACS Catal. 2012, 2, 1967−1975

clusters is $K'_{assoc.}$ = 6.1 ± 4.4 × $10^3$ $M^{-1.86}$. The $m'$ is 1.86 ± 0.15 (i.e., ca. 2), so that about 2 equiv of 1,10-phenanthroline per $Rh_4Cp^*_{2.4}Cl_4H_c$ are required to completely deactivate the $Rh_4$ catalyst—a result that makes physical sense, that is, that there are about 2 1,10-phenanthroline binding sites in the ligated, $Rh_4$ subnanometer cluster. Significantly, a $m'$ of about 2 is a physically more reasonable value than results from the literature-recommended linear treatment of the data and resultant $x_{intercept}$ value of 4.0 ± 0.4—and its implied about 4 equiv of 1,10-phenanthroline per total rhodium, about 16 equiv of 1,10-phenanthroline per $Rh_4$ cluster, Figure 2, vide supra.[12] The $m'$ about 2 value implies that, on average, one 1,10-phenanthroline binds one of the about two total vacant 1,10-phenanthroline coordination sites on the $Rh_4$ cluster, at least under the specific benzene hydrogenation catalysis conditions employed of 2-propanol at 100 °C and 50 atm initial $H_2$ pressure.[47,48]

**Quantitative Analysis of Recent Literature Poisoning Data: $CS_2$ Poisoning of Ammonia-Borane Dehydrocoupling at 25 °C Beginning with [Ru(cod)(cot)].** Zahmakıran and co-workers recently reported quantitative $CS_2$ poisoning data for ammonia-borane dehydrocoupling beginning with a [Ru(cod)(cot)] precatalyst.[49] Although TEM and zero contrast-TEM investigation of the resultant reaction mixture revealed the presence of agglomerated, about 60 nm Ru nanoparticles, quantitative poisoning experiments (using the $CS_2$ method we developed for nanoparticles in 2002[11]) showed that the addition (once the hydrogen evolution is 40% complete) of even 2 equiv of $CS_2$ per total Ru *did not completely poison the catalyst* (Supporting Information, Table SI-2); instead, about 13% of the initial activity still remained. Their quantitative $CS_2$ poisoning data are reproduced in Figure 4 and

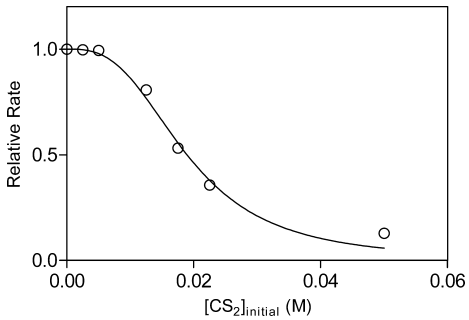



**Figure 4.** Curve-fit (solid line) of the reported[49] $CS_2$ poisoning data (○), Supporting Information, Table SI-2, by eq 5 herein, $R^2$ = 0.989.

caught our eye, since they are also slightly sigmoidal, suggesting that the use of a linear, strong-binding poison model is not appropriate but, instead, that the weak-binding kinetic model in Scheme 2 developed herein might be applicable and a better treatment of the data.

To test if Scheme 2 and eq 5 herein could fit their data, a curve-fit was carried out on the data in Figure 4 using the weak-binding poison model in Scheme 2 and its associated eq 5. A good fit to the data is seen, Figure 4, one that makes apparent the sigmoidal nature of the poisoning curve and, therefore, the apparent inappropriateness of the strong-binding, linear extrapolation fit approach in Scheme 1 for the analysis of this particular data. (Ex-post-facto checks on the weak-binding assumption are also provided as part of the Supporting Information.) The results from the poisoning curve-fitting yield a $m''$ = 2.9 ± 0.4, one a value consistent with the reported[49] $x_{intercept}$ of ≥2.0 (that ≥2.0 value being obtained, however, via drawing a tangent line to an incomplete set of the poisoning data, one where data at 0.0 and 0.2 equiv of $CS_2$ per total Ru appear to have been arbitrarily excluded; see Supporting Information, Figure SI-9, in ref 49). Hence, the fit in Figure 4 at least illustrates the need for and value of Scheme 2 and its associated eq 5 as a way to begin to think more rigorously about how one might account for nonlinear, sigmoidal poisoning curves that look to be weak-binding cases.

The authors see 0.6−2.2 nm particles by microscopy (once the hydrogen evolution was ca. 30% complete), but on the basis of the poisoning studies propose that the actual catalysis is by *subnanometer*, Ru clusters, the precise identity and nuclearity of which remain to be determined. The finding herein of $m''$ = 2.9 ± 0.4 (i.e., and not a value closer to 0.1 for example) is in general support of their conclusion, and is probably consistent with about $Ru_{\sim 4}$ clusters. However, in-operando spectroscopy[13] is needed to identify the dominant form(s) of Ru present under the reaction conditions (i.e., only if the subnanometer cluster is the dominant form of the Ru mass present can one say that the finding of $m''$ = 2.9 ± 0.4 strongly supports a subnanometer, $Ru_{\sim 4}$, say, cluster catalyst (vs for example a larger nanoparticle catalyst). This literature example again illustrates both the importance of quantitiative kinetic poisoning experiments in determining the true catalyst, as well as the value of the treatment in Scheme 2 and the equations herein for treating such nonlinear poisoning plot data in a more rigorous fashion.

**Comment on the Formulation, Units, and Use of the Strong-Binding Poison Scheme 1 and Equation 1, vs the Weak-Binding Poison Scheme 2 and Equation 5.** In Scheme 1 we have deliberately left the catalyst as the less specific "Rh(0) nanoclusters", and have not formulated Scheme 1 using the *on-average* $Rh(0)_{\sim 700}$ that were subsequently shown to be present. We have formulated Scheme 1 this more general "Rh(0) nanoclusters" way since, at least to start, most researchers using Scheme 1 (or Scheme 2) may not know the resulting speciation from their precatalyst, much less the actual catalyst(s). That is, Scheme 1 has been deliberately formulated on a per-total-Rh present basis, although it could have been written on with $Rh(0)_{\sim 700}$ (resulting, then and of course, in a different definition of the $K_{assoc.}$ with its different units). A comparison of Scheme 1 and its associated eq 1, vs Scheme 2 and its associated eq 5, reveals that a fundamental difference in the strong-binding poison case and associated eq 1 is that one must pick a form of the added precatalyst (e.g., total Rh(0) or $Rh(0)_{\sim 700}$, for example) to define the system and its resultant $K_{assoc.}$ and eq 1.

In the weak-binding poison case, Scheme 2, the total Rh present (nor any other formulation of the putative catalyst) does not appear in the resultant eq 5. In Scheme 2 we went ahead and deliberately formulated Scheme 2 and its resultant $K'_{assoc.}$ definition in terms of $Rh_4$ clusters, since their presence as ≥98% of the Rh mass is known from the in-operando spectroscopy.[12] As already noted, the $K'_{assoc.}$ of Scheme 2 and the $K_{assoc.}$ of Scheme 1 have different units and cannot, therefore, be directly compared.

For the literature $CS_2$ poisoning of the ammonia-borane dehydrocoupling reaction beginning with a [Ru(cod)(cot)] precatalyst, we deliberately did not write a separate scheme, past noting that the weak-binding poisoning model was used to analyze the poisoning data, and since the dominant form of Ru present is not known for certain. However, from analyzing the

poisoning data and the $m'' = 2.9 \pm 0.4$ which resulted, one good hypothesis for future investigation is a version of Scheme 2 with $Ru_{\sim 4}$ as one possibility for the kinetically dominant catalyst.

In short, researchers using versions of Schemes 1 or 2 will want to decide how to best define analogous schemes for their own specific systems and poisoning studies. We recommend that poisoning schemes and resultant $K_{assoc.}$ (i.e., or $K'_{assoc.}$) values be defined as specifically as possible, such as in Scheme 2, but we illustrate herein both cases via Schemes 1 and 2 to more broadly cover the general cases and level of knowledge about the true catalyst that one can expect. In all cases, a more detailed interpretation of catalyst poisoning data will require a knowledge of at least the average or dominant speciation of the system, as provided for the $Rh(0)_{\sim 700}$ and $Rh_4$ strong- and weak-binding poison cases, respectively, herein.

## CONCLUSIONS

Analysis of 1,10-phenanthroline quantitative poisoning kinetic experiments, for Rh(0) nanoparticles as well as $Rh_4$ clusters undergoing benzene hydrogenation reaction at 100 °C and 50 atm initial $H_2$ pressure, led to several insights, including:

(i) A strong-binding model for 1,10-phenanthroline attachment to the Rh(0) nanoparticles can account for the observed catalyst poisoning data, and in a closer, more rigorous look at the data. A minimalist, strong-binding mechanistic model, Scheme 1, clarifies the common practice[1,11] of drawing a tangent to the often at least somewhat curved poisoning plot of the relative rate vs equiv of poison/equiv of total metal present. The $x_{intercept}$ of such classical treatments of the data does in fact give the total number of equivalents of poison neded to deactivate completely the catalyst, with $x_{intercept} = m$ of the strong-binding mechanistic model, Scheme 1, defined in that case on a per total Rh(0) present. In addition, a correspondence with eq 1 from Scheme 1 and the historical $k_c = k_0(1 - \propto c)$ equation used to analyze poisoning data since the 1950s was, while perhaps obvious at least in hindsight, clarified and mathematically equated. The data were then used to see what limit resulted for $K_{assoc.}$ in the strong-binding case, and the $m$ value and the average size of the nanoparticles were used to estimate the fraction of surface catalytically active sites.

(ii) Second, a weak-binding model for 1,10-phenanthroline attachment to $Rh_4$ subnanometer clusters was shown to account for that observed catalyst poisoning data. The initially nonlinear, slightly sigmoidal poisoning curve was shown to very nicely and quantitatively fit the data, yielding physically reasonable $K'_{assoc.}$ and $m'$ values. The $m'$ value was then used to provide a probably good estimate of the number of 1,10-phenanthroline binding sites (two) on the $Rh_4$ cluster catalyst of average composition $Rh_4Cp^*_{2.4}Cl_4H_c$.[12] In the weak-binding poison case, the formulation of Scheme 2 was given in terms of the more desirable formulation of the specific dominant species shown to be present by in-operando XAFS, namely, $Rh_4$ subnanometer clusters of average composition $Rh_4Cp^*_{2.4}Cl_4H_c$.

(iii) Third, an example of interesting, recent $CS_2$-based catalyst poisoining data from the literature[49] was analyzed and shown to be quantitatively accounted for by the weak-binding poison kinetic model developed herein. The results provide credence to both the broader applicability of the weak-binding model as well as the value[12] of quantitative catalyst poisoning experiments in correctly and rapidly identifying the true catalyst in a given system. The resultant $m'' = 2.9 \pm 0.4$ suggests a version of Scheme 2, with $Ru_{\sim 4}$ as one possibility for the kinetically dominant catalyst, as one hypothesis for future studies of the identity of the true catalyst in that interesting amine-borane dehydrocoupling catalysis system.

(iv) The results presented herein also are important in that they fortify our recent conclusion[12] that $Rh_4Cp^*_{2.4}Cl_4H_c$ subnanometer clusters are the true catalyst in benzene hydrogenation beginning with Maitlis' classic system discovered some 35 years ago[50] of $[RhCp^*Cl_2]_2$ and at 100 °C and 50 atm initial $H_2$ pressure. Specifically, the results herein disprove the alternative hypothesis we raised[12] of "...1,10-phenanthroline poison could be bound first by the $Rh_4$ clusters, with no poisoning reaching the (in this case hypothesized, true) Rh(0) catalyst until the 1,10-phenanthroline binding capacity of the $Rh_4$ clusters had been saturated."[12] The results herein rule out this possibility since the Rh(0) nanoclusters have the higher affinity for the 1,10-phenanthroline poison than do the heavily ligated, apparently sterically more congested, $Rh_4Cp^*_{2.4}Cl_4H_c$ subnanometer clusters.

(v) Finally, the results herein and those of others[26,51] provide additional evidence for our assertion[12] that quantitative catalyst poisoning experiments can provide some of the strongest, often necessary, evidence for correctly identifying the true catalyst in multiple types of catalytic reactions, be they the benzene hydrogenations[12] and amine-borane dehydrocoupling catalysis data[49] treated herein or the transfer hydrogenation of ketones[51] or CO oxidation catalysts[26] reported by others.

## ASSOCIATED CONTENT

### Supporting Information

Derivation of the equations for the strong-binding poison case; analysis of the sigmoidal quantitative 1,10-phenanthroline poisoning plot for $Rh_4$ subnanometer clusters; ex-post-facto confirmation of $[B'_0] \gg m'[P']$ with the $K'_{assoc.}$ and $m'$ values obtained via nonlinear least-squares fitting of the poisoning data; comparison of $K'_{assoc.}$ values obtained by the controls of using the now available $[B'_0] - m'[P']$ values and fit the poisoning data; additional 1,10-phenanthroline poisoning experiment results with Rh(0) nanoparticles; analysis of the recently published quantitative $CS_2$ poisoning of ammonia-borane dehydrocoupling reaction beginning with [Ru(cod)-(cot)] at 25 °C, including ex-post-facto justification of the weak-binding assumption. This material is available free of charge via the Internet at http://pubs.acs.org.

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### Funding

Financial support was provided by the Department of Energy, Grant DE-FG02-03ER15453.

### Notes

The authors declare no competing financial interest.

## ACKNOWLEDGMENTS

Authors would like to thank Finke-group members Jordan Stracke, Dr. Joseph Mondloch, and Will Laxson for valuable discussions during the preparation of the manuscript.

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(47) Note that following the original recipe for catalysis in this system,[12] 15 equiv of $Et_3N$ are added per initial Rh employed. One role of the added $Et_3N$ is to capture HCl formation produced from heterolytic $H_2$ activation.[48] Assuming our previously proposed[12] stoichiometry for the formation of $Rh_4$ clusters of $2[RhCp^*Cl_2]_2 + 4Et_3N + (5.6 + c)/2\ H_2 + Rh_4Cp^*_{2.4}Cl_4H_c + 4Et_3NH^+Cl^- + 1.6(Cp^*\text{-}H)$, then only 1 equiv of $Et_3N$ per equiv of Rh will form $Et_3NH^+Cl^-$, leaving 14 equiv of $Et_3N$ "free" and thus itself a "potential" ligand/poison.[10] However, and as a control to test for the rate effect of the added 15 equiv of $Et_3N$, a Standard Conditions benzene hydrogenation with $[RhCp^*Cl_2]_2$ was performed as detailed elsewhere[12] *without any added* $Et_3N$ followed by a subsequent benzene hydrogenation with the fully evolved catalyst (i.e., with $Rh_4$ clusters). That control yielded the same kinetics within ±15% experimental error as reported previously; see Figure 1 and Figure 7, respectively, elsewhere,[12] implying a minimal rate effect of the added 15 equiv of $Et_3N$. In addition, the 1,10-phenanthroline poisoning data analyzed herein are *relative rates*, so that any small effect of the 15 equiv of $Et_3N$ should cancel out (i.e., any small effects of the 15 equiv of $Et_3N$ are also in the initial rate of benzene hydrogenation beginning with $Rh_4$ clusters without any added 1,10-phenanthroline, Supporting Information, Figure SI-3a). In short, the evidence would seem to rule out the

hypothesis that the 15 equiv of $Et_3N$, that are part of the original recipe for this system,[12] are somehow distorting the analysis or interpretation of the 1,10-phenanthroline poisoning experiments reported herein.

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