Department of Chemistry
Cumulative Examinations
April 29, 2006

You may choose to answer any exam from any area covered in the examination booklet. Each exam may contain multiple parts. You may answer more than one exam but each exam is scored separately and is treated as an individual examination result. Thus, answering parts of two exams with a score of 50% *would not* yield a 100% grade for this cumulative exam. Instead you would receive 50% on each examination attempted.

This booklet contains ***five*** examinations.

1) Analytical Cumulative Examination, Page 1
2) Biochemistry Cumulative Examination, Pages 2-3
3) Inorganic Cumulative Examination, Page 4
4) Organic Cumulative Examination, Pages 5-8
5) Physical Cumulative Examination, Pages 9-10

On your examination booklet:

1) Print your student ID number.
2) Print this Exam Booklet number: ___________________
3) Print the question number you are answering.
4) Print the Exam Date.

Do not write your name anywhere on the examination booklet. Each exam will be scored anonymously. If you attempt more than one exam, *you must use a separate examination booklet for each examination*.

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PURDUE
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Cume Analytical Chemistry
April 29, 2006

Aspects of Quantitative Analysis: Liquid-liquid Extractions

1. The *distribution constant, K_D*, is defined as the ratio of the species concentrations of a given analyte between the organic and aqueous phase. This is an equilibrium constant.

a. If K_D is 9.8 and the aqueous concentration of the analyte at equilibrium is 3.2 μM, predict the equilibrium concentration in the organic phase.

2. For situations where the heterogeneous extraction equilibrium is coupled to homogeneous equilibria in some of the liquid phases, the overall extraction is described with the *distribution ratio, D*. It is defined as the ratio of the analytical (total) concentrations of a solute in the organic and aqueous phases at equilibrium.

a. Is the distribution ratio, D, an equilibrium constant? Please discuss.

b. Write down an expression for D for a weak acid HA as a function of the distribution constant, K_D, the pK_a in the water phase and the pH. Assume the acid does not dissociate in the organic phase.

c. Calculate K_D if log D = -3.2, pK_a = 4.5 and pH = 6.5.

3. To facilitate extraction of metal ions into an organic phase, an excess of metal chelator can be added to the organic phase. Consider, for example, the chelator 8-hydroxyquinoline, HQ. It interacts with divalent metal ions, M^{2+}, to form the complex MQ_2 in the organic phase. You can neglect the formation of this complex in the aqueous phase.

a. Write down the overall balanced extraction equilibrium reaction and associated equilibrium constant expression for the extraction of M^{2+} by HQ to form MQ_2.

b. Write down the appropriate relationship for D as a function of the overall equilibrium constant and the pH.

4. *Optical ion sensors* can be described in analogy to liquid-liquid extractions. In one example, the oil-like sensing phase contains an electrically neutral receptor L for the analyte ion M^+ and an electrically neutral receptor/indicator Ind for the reference hydrogen ion H^+.

a. Think about and propose other components needed in the sensing phase and sketch how the selective extraction of M^+ may be coupled to the extraction of the reference ion H^+ to make this sensor work (in absorbance mode).

b. Write down the appropriate expression for the overall extraction equilibrium and the equilibrium constant.

c. Discuss how the extraction is influenced by pH and by the $pK_a$of the indicator, Ind (pK_a defined for the organic phase).

Biochemistry Division Cume Membranes and Membrane Proteins
April 29, 2006

1. Define "critical micellar concentration" of a detergent or lipid. What role does the CMC of a detergent play in the purification of a membrane-bound protein and it's reconstitution into artificial membrane vesicles? What type of CMC for a detergent is preferred in a typical reconstitution experiment, and why?

2. Define a lipid raft. What is the size and composition of a raft. What are some proposed functional properties of rafts? How would you design an experiment to test the importance of the raft in any one of these functional properties?

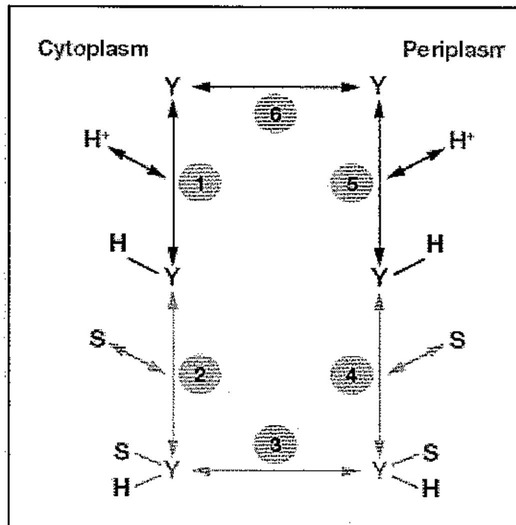
3. A fluorescent group is attached to a component of a lipid bilayer. An intense laser pulse is focused on a very small area to permanently destroy (bleach) the fluorophore in that area. Fluorescence microscopy can be used to monitor what happens after the laser pulse.

A. Describe what would be observed after photobleaching is complete and
give an approximate time frame for these events.

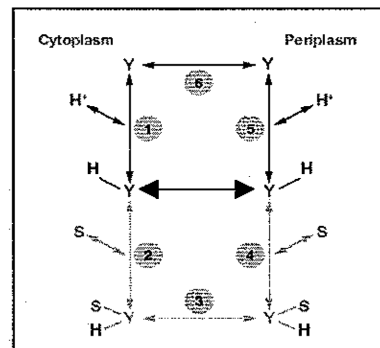
B. What can be learned about the membrane and its components from this
experiment?

4. At times, it is biologically important for a lipid in a membrane to move from one layer of the lipid bilayer to the other. Discuss two possible mechanism by which this could occur and the energetics involved in the process.

5. The following scheme describes a common mechanism for transport in a prokaryotic cell membrane. In this scheme, Y represents a transport protein, H, a proton and S a transport substrate. In the transport cycle, (which is fully reversible), a proton binds to Y in the cytoplasm (step 1), then substrate binds (step 2). With both H and S bound, Y undergoes a conformational change that exposes ligand-binding sites only to the periplasm (step 3). Substrate dissociates (step 4), then proton dissociates (step 5) and Y returns to it's original conformation, with ligand-binding sites exposed only to the cytoplasm (step 6).

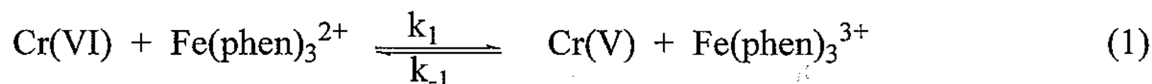


A. What type of transport mechanism is being described by this scheme?
B. Does this six-step mechanism offer any advantage to the cell as opposed to a simpler scheme in which S alone binds Y and triggers conformational change?
C. What would happen to the transporter if step 6 could not occur?
D. How would the addition of a double-headed arrow across the center (see new Fig) affect the mechanism of transport?



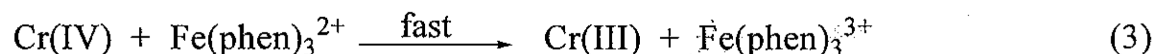
INORGANIC CHEMISTRY CUMULATIVE EXAM
April 29, 2006

1. In a recent paper by Du and Espenson, *Inorg. Chem.* **2006**, *45*, 1053–1058, the following mechanism is discussed for the stepwise reduction of Cr(VI) by tris(1,10-phenanthroline)iron(II) ions.



$$Cr(VI) + Fe(phen)_3^{2+} \underset{k_{-1}}{\overset{k_1}{\rightleftharpoons}} Cr(V) + Fe(phen)_3^{3+} \quad (1)$$

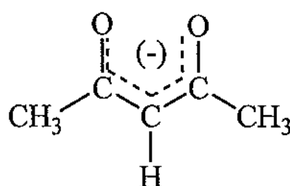
$$Cr(V) + Fe(phen)_3^{2+} \xrightarrow{k_2} Cr(IV) + Fe(phen)_3^{3+} \quad (2)$$



$$Cr(IV) + Fe(phen)_3^{2+} \xrightarrow{fast} Cr(III) + Fe(phen)_3^{3+} \quad (3)$$

(a) Derive the rate expression for the rate of loss of $Fe(phen)_3^{2+}$ if reactions (1) and (2) have Cr(V) as a steady-state species and reaction (3) is fast.
(b) Show what is meant by the steady-state approximation for this system.
(c) Give the chemical equation for the overall reaction.

2. **(a)** The structure of $acac^-$ is:



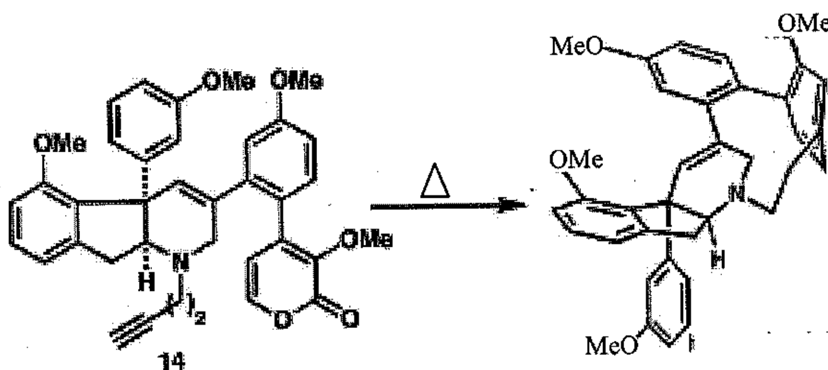
Show two possible products for the reaction of H_3O^+ with $acac^-$. Discuss which reaction is faster and why. Which product is more stable? Show the reactions needed for the initial protonated product to convert to the more stable product.
(b) In general-acid-assisted reactions, how does the acidity of the acids affect the rate of proton transfer? Show the type of correlation to be expected and specify the range of slopes possible for Brønsted plots.

3. Outline the stepwise chemical reactions that take place during an alkene ($H_2C{=}CHR$) hydrogenation with Wilkinson's catalyst, $(Ph_3P)_3RhCl$. Specify the steps that correspond to oxidative addition, reductive elimination, and hydride migration. Show structures for all intermediates.

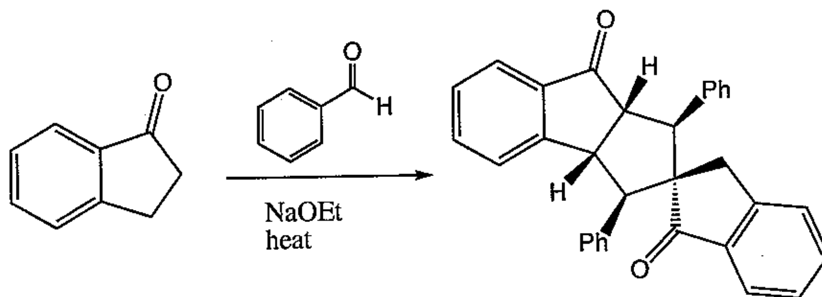
Organic Cumulative Exam – April 2006

Please answer the following questions. The articles from problems 3 and 4 are attached.

1. Please provide a mechanism for the following reaction.
   JACS, Vol. 128, 3908-3909, 2006.



2. Please provide the mechanism for the following reaction.
   JOC, 2006, ASAP



3. Please answer the following questions based on article I attached. [JACS 128, 386 (2006)]
   a. Draw a schematic that accounts for the cell-based entry of the His-tagged GFP promoted by compound **1**.
   b. What was the optimum concentration of compound **1** to facilitate His-tagged GFP cell entry? Why might further addition of **1** result in a decrease in GFP entry? Why might pre-mixing all of the reagents results in a decrease in GFP entry?
   c. In panel B from Figure 2, the cells were washed with NTA prior to microscopy. What was the point of this procedure?

4. Please answer the following questions based on article II attached. [JACS 127, 4128 (2005)]
   a. Which polymer(s) was/were the most potent antimicrobial agent? Estimate the MIC value(s).
   b. Which polymer(s) was/were the least toxic?
   c. Which polymer(s) has/have a window of efficacy in which they will be selectively toxic to bacteria? What % MP_{Bu} would this occur at?
   d. What effect do molecular weight and hydrophobicity have on the antimicrobial activity of these polymers?

Article I

JACS COMMUNICATIONS

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Synthesis of an Artificial Cell Surface Receptor that Enables Oligohistidine Affinity Tags to Function as Metal-Dependent Cell-Penetrating Peptides

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Cell-penetrating peptides and proteins (CPPs) have emerged as important new tools for the delivery of impermeable molecules into living mammalian cells. These delivery systems have been constructed from basic segments of HIV Tat,[1] oligoguanidine-containing peptoids,[2] β-peptides,[3] and other oligocationic motifs.[4] The mechanism of cellular penetration by CPPs is primarily thought to involve endocytosis,[5] particularly with larger cargo,[6] but varies depending on the delivery system and the cell type.[7]

Oligohistidine peptides are often fused to proteins to provide affinity tags ("His tags") that facilitate protein purification. To enable these peptides to function as CPPs, we synthesized an artificial cell surface receptor (1) comprising the plasma membrane anchor *N*-alkyl-3β-cholesterylamine linked to the metal chelator nitrilotriacetic acid (NTA). *N*-Alkyl-3β-cholesterylamine derivatives can function as prosthetic molecules active on the surface of living mammalian cells because this steroid can insert into cellular plasma membranes, project linked headgroups from the cell surface, and rapidly cycle between the plasma membrane and intracellular endosomes, similar to many naturally occurring cell surface receptors.[8,9] The NTA motif binds tightly to nickel, cobalt, copper, and zinc dications, and immobilized metal chelate chromatography (IMAC) with NTA-linked supports is widely used to purify proteins fused to His tags.[10] Other lipids[11,12] and polymers[13] linked to NTA headgroups have also been reported.

Receptor **1** was synthesized from 3β-cholesterylamine (**2**)[9] as shown in Scheme 1. Alkylation of **2** with ethyl 5-bromovalerate, followed by Boc protection to afford **3**, provided an improved route to protected *N*-alkyl derivatives of the membrane anchor. Addition of a linker shown to increase the population of these compounds on the cell surface,[9] acylation with a protected NTA derivative, and final deprotection afforded **1** in 47% overall yield.

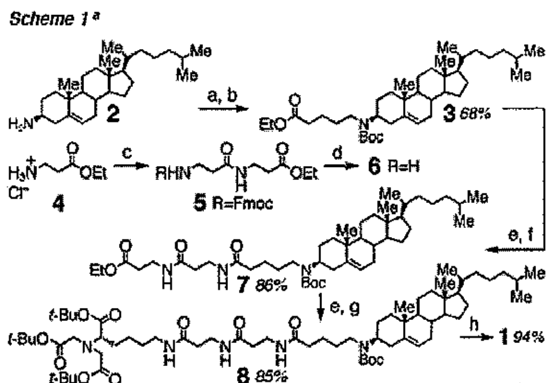
As representative His-tagged proteins, we overexpressed in *E. coli* the monomeric green fluorescent protein AcGFP fused to C-terminal $(His)_6$ and $(His)_{10}$ peptides and purified these proteins by IMAC. The $(His)_{10}$ peptide binds Ni−NTA complexes ~6-fold more tightly than $(His)_6$. For example, $(His)_6$ and $(His)_{10}$ peptides fused to $5HT_3R$ serotonin receptors exhibit affinities for Ni−NTA derivatives of $K_d = 1.05$ μM and $K_d = 166$ nM, respectively.[14]

To qualitatively examine the ability of receptor **1** to mediate the cellular uptake of His-tagged proteins, human Jurkat T-lymphocytes were treated with **1** (10 μM) for 1 h to load this compound into the outer leaflet of the cellular plasma membrane. These cells were washed and subsequently treated with a solution of $AcGFP(His)_{10}$ (3.2 μM) and $Ni(OAc)_2$ (100 μM) for an additional 4 h. As shown in Figure 2 (panel A), examination of these cells by confocal laser scanning microscopy revealed fluorescent protein both on cell surface and in defined intracellular compartments. Washing the cells with disodium NTA (400 μM)

Delivery of AcGFP-$(His)_6$ under these conditions provided similar results, but the cells were 4-fold less fluorescent (Figure 3, panel D). The fate of internalized $AcGFP(His)_{10}$ was examined by coadministration with DiI-loaded low-density lipoprotein (LDL), a protein internalized by receptor-mediated endocytosis, as a red fluorescent marker of endosomes and lysosomes.[15] Substantial colocalization of red and green fluorophores was observed under these conditions (Supporting Information). This delivery to endosomes and lysosomes represents a common destination of proteins fused to CPPs.[16]

Quantitative analysis by flow cytometry of synthetic receptor-mediated uptake is shown in Figure 3. Intracellular delivery of $AcGFP(His)_{10}$ >600-fold above basal levels of endocytosis was achieved by preloading the cellular plasma membrane with receptor **1** (10 μM) for 1 h followed by addition of the protein (3.2 μM) and $Ni(OAc)_2$ (100 μM). Higher receptor concentrations, addition

Scheme 1 [a]



[a] Reagents and conditions: (a) ethyl 5-bromovalerate, K_2CO_3, DMF, 60 °C; (b) $(Boc)_2O$, DIEA, CH_2Cl_2; (c) *N*-Fmoc-β-Ala, EDC, HOBt, DIEA, CH_2Cl_2; (d) piperidine, DMF; (e) $LiOH \cdot H_2O$, MeOH/THF/H_2O (3:2:1); (f) **6**, EDC, HOBt, CH_2Cl_2; (g) N^α,N^α-bis[(*tert*-butoxycarbonyl)methyl]-L-Lys *tert*-butyl ester, EDC, HOBt, CH_2Cl_2; (h) TFA, CH_2Cl_2.

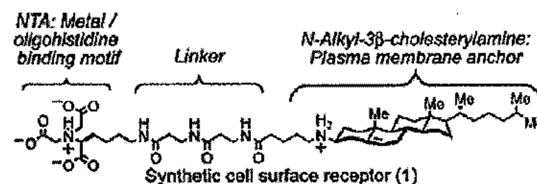


Figure 1. Top: Structure of the synthetic cell surface receptor (1) that binds metals and His tags. Bottom: Strategy for delivery of His-tagged proteins into mammalian cells by synthetic receptor-mediated endocytosis.

10.1021/ja056126j CCC: $33.50 © 2006 American Chemical Society

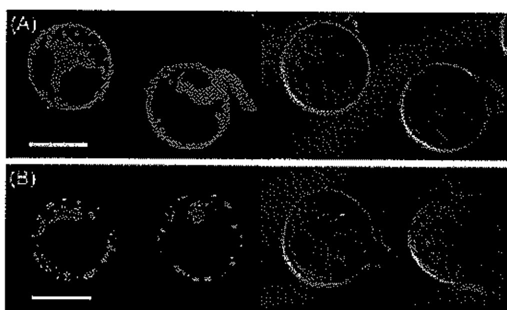


Figure 2. Confocal laser scanning (left) and differential interference contrast (right) micrographs of living Jurkat lymphocytes. Cellular plasma membranes were preloaded with receptor 1 (10 μM) for 1 h at 37 °C, cells were washed with fresh media, and media containing AcGFP(His)$_{10}$ (3.2 μM) and Ni(OAc)$_2$ (100 μM) was added for an additional 4 h at 37 °C. Cells shown in panel A were imaged immediately after this treatment. Cells shown in panel B were washed with NTA (400 μM, 30 min) prior to microscopy . Scale bar = 10 μm.

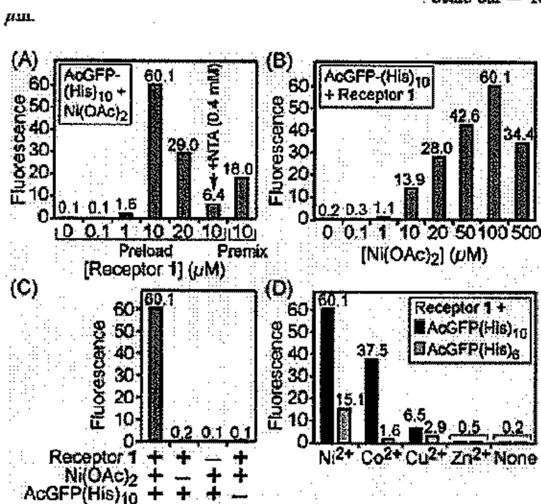


Figure 3. Analysis of cellular uptake of His-tagged AcGFP proteins by flow cytometry. Each bar represents the average fluorescence of 10 000 cells. Unless otherwise noted, cellular plasma membranes of Jurkat lymphocytes were preloaded with receptor 1 (10 μM) for 1 h at 37 °C, cells were washed with fresh media, and His-tagged AcGFP (3.2 μM)/metal diacetate solution (100 μM) was added for 4 h at 37 °C. Prior to analysis, cells were washed with NTA (400 μM, 30 min) in PBS (pH 7.4) to remove bound surface protein. Panel A: Dose dependence of receptor-mediated uptake. Premix conditions: A solution containing receptor 1, Ni(OAc)$_2$, and AcGFP(His)$_{10}$ was added to cells for 4 h. Panel B: Dependence on [Ni(OAc)$_2$]. Panel C: Omission control experiments. Panel D: Uptake of (His)$_6$ and (His)$_{10}$ fusion proteins promoted by different metal diacetates.

of disodium NTA as a competitor, or premixing the receptor with the protein and metal resulted in lower levels of uptake (panel A). Preloading the plasma membrane may facilitate uptake due to the high surface density of immobilized metal chelate.[17,18] Significantly higher or lower concentrations of Ni(OAc)$_2$ were less effective mediators of uptake (panel B), and omission control experiments confirmed the importance of all three components (panel C). Comparison of diacetates of Ni, Co, Cu, and Zn revealed that Ni(OAc)$_2$ was the most effective metal for delivery of both (His)$_6$ and (His)$_{10}$ fusion proteins.

Consistent with the mechanism of synthetic receptor-mediated endocytosis,[9] uptake of His-tagged AcGFP proteins was blocked by cooling cells to 4 °C (data provided in the Supporting Information). These conditions enabled quantification of the number of synthetic receptors on the cell surface. Jurkat lymphocytes were treated with 1 (10 μM) for 1 h at 37 °C, the cells were washed, chilled to 4 °C, and excess AcGFP(His)$_{10}$ (19 μM) and Ni(OAc)$_2$ (100 μM) were added to saturate the surface-bound NTA groups. Comparison of these cells with fluorescent bead standards by flow cytometry revealed an average of ~45,000,000 synthetic receptors per cell surface. Previous studies of related compounds suggest that a similar population of synthetic receptors resides in intracellular endosomes.[9]

Heavy metals, such as Ni^{2+}, exhibit dose-dependent toxicity to biological systems. To examine the toxicity of this metal acetate and protein delivery, cellular viability was examined by flow cytometry. In these experiments, Jurkat lymphocytes were treated with receptor 1 (10 μM, 1 h), followed by AcGFP(His)$_{10}$ (3.2 μM), and Ni(OAc)$_2$ (100 μM, 4 h) to enhance protein uptake by 600-fold. Cells were washed with disodium NTA to remove cell surface protein and were cultured for an additional 48 h. Cellular viability was determined by light scattering and staining the nuclei of membrane-compromised dead cells with the fluorophore propidium iodide. Under these conditions, 97% of the cells were viable, compared with 99% of untreated controls, and no effects on cellular morphology were observed. Other studies have similarly reported low toxicity of nickel salts at $\leq$160 μM in cell culture.[19] By enabling common oligohistidine affinity tags to function as cell-penetrating peptides, synthetic receptor 1 provides a potentially versatile new probe of cellular biology.

Acknowledgment. We thank the NIH (R01-CA83831) for financial support.

Supporting Information Available: Supporting figures (S1−S3), compound characterization data, and experimental procedures. This material is available free of charge via the Internet at http://pubs.acs.org.

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Article II

JACS COMMUNICATIONS

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Amphiphilic Polymethacrylate Derivatives as Antimicrobial Agents

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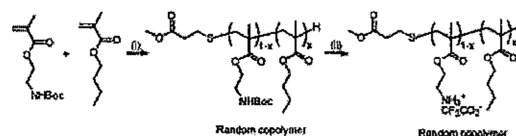
The membrane-disrupting ability of amphiphilic synthetic polymers has been utilized in preparing chemical disinfectants and biocides.[1] A number of polymeric disinfectants have been prepared using conventional synthetic polymers, including poly(vinyl pyridine)s,[2] poly(vinyl alcohol)s,[3] polyacrylates,[4] and polystyrenes.[5] Their amphiphilic structures disrupt cell membranes,[1] causing breakdown of the transmembrane potential, leakage of cytoplasmic contents, and ultimately cell death. The cooperative action inherent in polymeric structures enhances this disruption mechanism as compared to small amphiphilic molecules, such as surfactants.[6] However, one of the major drawbacks of polymeric disinfectants is a lack of selectivity for bacterial over human cells, limiting their clinical and medicinal utility.

In this report, we take steps toward the development of nontoxic antimicrobial synthetic polymers in our investigation of the structure–activity relationship of amphiphilic polymethacrylate derivatives as measured via antimicrobial and hemolytic activities. Free radical copolymerizations of *N*-(*tert*-butoxycarbonyl)aminoethyl methacrylate and butyl methacrylate (BMA) were conducted using mole percentages of BMA from 0 to 60% in the presence of methyl 3-mercaptopropionate (MMP) to give a precursor polymer protected with *tert*-butoxycarbonyl (Boc) groups (Scheme 1). In this polymerization, MMP served as a chain transfer agent to control the degree of polymerization (DP)[7] and allowed us to prepare low molecular weight (MW) polymers with relatively high yields, avoiding the necessity of time-intensive fractionation of polymers by column chromatography. The subsequent treatment of the Boc-protected polymer with TFA afforded the desired cationic random copolymer. Natural host defense peptides are believed to selectively target bacterial cells while remaining nontoxic to the host due to preferable charge interactions between the dense population of negatively charged lipids on bacterial cell surfaces and the cationic side chains of the peptides;[8,9] we have similarly incorporated cationic functionality into the polymer framework to improve selectivity. By alternating MMP concentrations, we obtained a series of polymers of three different MW ranges displaying a wide range of mole percentages of BMA (MP_{Bu}) (0–60%) (Table 1).[10]

Antimicrobial activity of the polymers was tested using turbidity-based assays in Mueller–Hinton broth with *Escherichia coli* D31, and the minimum inhibitory concentration (MIC) was determined as the lowest polymer concentration to completely inhibit bacterial growth after an 18 h incubation period at 37 °C.

Scheme 1. Synthesis of Amphiphilic Polymethacrylate Derivatives[a]



[a] Conditions: (i) methyl 3-mercaptopropionate, AIBN, acetonitrile, 60 °C, overnight; (ii) neat TFA, rt, 1 h.

Table 1. Characterization of Amphiphilic Polymethacrylates

series	[MMP]/[monomers][a]	MP_{Bu}[b]	DP[b]	MW range[c]
1	0.05	0–57	32–46	7900–10100
2	0.10	0–53	19–31	4500–6000
3	0.50	0–47	5–9	1300–1900

[a] Concentration ratio of MMP to total amount of monomers in polymerizations. [b] Determined by ^{1}H NMR. [c] Calculated from MP_{Bu} and DP.

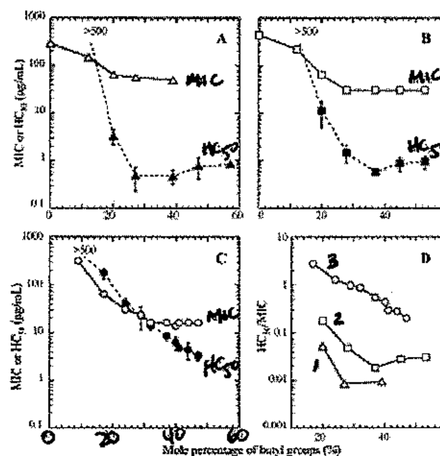


Figure 1. Antimicrobial and hemolytic activities of polymethacrylate derivatives: (A) polymer series 1; (B) 2; and (C) 3. MICs and HC_{50}s presented as opened and closed marks, respectively. (D) Selectivity index (HC_{50}/MIC).

Mole % of butyl groups (x-axis)

Important features of polymers useful as disinfectants are not only their antimicrobial activity but also the lack of toxicity to human cells, particularly for medical and clinical utility. Toward this end, their lytic activity against human red blood cells (hemolytic activity) was evaluated as HC_{50} for each series of polymers, which is the polymer concentration necessary for 50% lysis of cells. In

Physical
Cumulative Exam
April 29, 2006

1. Consider a binary mixture composed of benzene (B) and camphor (C). B melts at 5.5°C and boils at 80.1°C. C melts at T = 179°C and decomposes without boiling at high temperatures.

a. Assume the solution has a mole fraction of C $x_C = 0.27$ and is in equilibrium with its vapor at 80.1°C. Compute the vapor pressure P in atmospheres of the solution at 80.1°C using Raoult's law.

The rest of the problem will concern **three-phase** states of the B plus C solution comprised of the vapor (v) phase in equilibrium with a single liquid (l) and a single solid(s) phase.

b. Using the Gibbs phase rule F = C – P +2 determine how many intensive variables F may be independently varied without disturbing the three-phase equilibrium.

c. The Gibbs free energy of the mixture may be written as

$$G = G_v(T,P,n_{B_v},n_{C_v}) + G_l(T,P,n_{B_l},n_{C_l}) + G_s(T,P,n_{B_s},n_{C_s}) \cdot \quad (1.1)$$

where G_v is the Gibbs free energy of the vapor phase, and so on.

(i) Show from Eq. (1.1) that for the three phases to be in equilibrium at fixed T,P

$$\mu_{B_v} dn_{B_v} + \mu_{C_v} dn_{C_v} + \mu_{B_l} dn_{B_l} + \mu_{c_l} dn_{c_l} + \mu_{B_s} dn_{B_s} + \mu_{C_s} dn_{C_s} = 0 \cdot \quad (1.2)$$

(ii) Show that

$$dn_B = dn_{B_v} + dn_{B_l} + dn_{B_s} = 0 \text{ and } dn_C = dn_{C_v} + dn_{C_l} + dn_{C_s} = 0 \cdot \quad (1.3)$$

(iii) From Eqs. (1.2) and (1.3) show that a sufficient condition for three-phase equilibrium is

$$\mu_{B_v} = \mu_{B_l} = \mu_{B_s} \text{ and } \mu_{c_v} = \mu_{C_l} = \mu_{C_s} \cdot \quad (1.4)$$

d. Using Eq. (1.4) do the following
   (i) Write down the equilibrium equation for the solubility of C assuming the s phase is pure C. Explain your reasoning.

   (ii) Write down the equilibrium equation for the boiling point elevation of B assuming the v phase is pure B. Explain your reasoning.

e. Rewriting Eqs. (1.4) in more detail as

$$\mu_{B_v}(T,P,x_{B_v}) = \mu_{B_l}(T,P,x_{B_l}) \tag{1.5a}$$
$$\mu_{B_v}(T,P,x_{B_v}) = \mu_{B_s}(T,P,x_{B_s}) \tag{1.5b}$$
$$\mu_{B_l}(T,P,x_{B_l}) = \mu_{B_s}(T,P,x_{B_s}) \tag{1.5c}$$

and

$$\mu_{C_v}(T,P,x_{C_v}) = \mu_{C_l}(T,P,x_{C_l}) \tag{1.5d}$$
$$\mu_{C_v}(T,P,x_{C_v}) = \mu_{C_s}(T,P,x_{C_s}) \tag{1.5e}$$
$$\mu_{C_l}(T,P,x_{C_l}) = \mu_{C_s}(T,P,x_{C_s}) \tag{1.5f}$$

Derive the result from F you found from the Gibbs phase rule in Part b. Explain your reasoning.

Periodic Classification of the Elements

I A	II A	III B	IV B	V B	VI B	VII B	VIII	VIII	VIII	I B	II B	III A	IV A	V A	VI A	VII A	O
1 H 1.00797																	2 He 4.0026
3 Li 6.939	4 Be 9.0122											5 B 10.811	6 C 12.01115	7 N 14.0067	8 O 15.9994	9 F 18.9984	10 Ne 20.183
11 Na 22.9898	12 Mg 24.312											13 Al 26.9815	14 Si 28.086	15 P 30.9738	16 S 32.064	17 Cl 35.453	18 Ar 39.948
19 K 39.102	20 Ca 40.08	21 Sc 44.956	22 Ti 47.90	23 V 50.942	24 Cr 51.996	25 Mn 54.9380	26 Fe 55.847	27 Co 58.9332	28 Ni 58.71	29 Cu 63.54	30 Zn 65.37	31 Ga 69.72	32 Ge 72.59	33 As 74.9216	34 Se 78.96	35 Br 79.909	36 Kr 83.80
37 Rb 85.47	38 Sr 87.62	39 Y 88.905	40 Zr 91.22	41 Nb 92.906	42 Mo 95.94	43 Tc (99)	44 Ru 101.07	45 Rh 102.903	46 Pd 106.4	47 Ag 107.870	48 Cd 112.40	49 In 114.82	50 Sn 118.69	51 Sb 121.75	52 Te 127.60	53 I 126.9044	54 Xe 131.30
55 Cs 132.905	56 Ba 137.34	57 La* 138.91	72 Hf 178.49	73 Ta 180.948	74 W 183.85	75 Re 186.2	76 Os 190.2	77 Ir 192.2	78 Pt 195.09	79 Au 196.967	80 Hg 200.59	81 Tl 204.37	82 Pb 207.19	83 Bi 208.980	84 Po (210)	85 At (210)	86 Rn (222)
87 Fr (223)	88 Ra (226)	89 Ac† (227)															

***Lanthanides**	58 Ce 140.12	59 Pr 140.907	60 Nd 144.24	61 Pm (147)	62 Sm 150.35	63 Eu 151.96	64 Gd 157.25	65 Tb 158.924	66 Dy 162.50	67 Ho 164.930	68 Er 167.26	69 Tm 168.934	70 Yb 173.04	71 Lu 174.97
†Actinides	90 Th 232.038	91 Pa (231)	92 U 238.03	93 Np (237)	94 Pu (242)	95 Am (243)	96 Cm (247)	97 Bk (247)	98 Cf (249)	99 Es (254)	100 Fm (253)	101 Md (256)	102 No (256)	103 Lw (257)

(Numbers in parentheses are the mass numbers of the most stable isotopes.)