Investigation of PFOA Enrichment Mechanisms in Sea Spray Aerosol Proxy Surfaces and Potential Health Implications

Honors Research Thesis

Presented in Partial Fulfillment of the Requirements for Graduation "with Honors Research Distinction in Public Health" in the undergraduate colleges at The Ohio State University

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ABSTRACT

Perfluoroalkyl substances are man-made chemicals that have been widely used in industrial processes and military operations since World War II. While the exact toxicology of these substances has not been deciphered, perfluorooctanoic acid (PFOA) has been linked to liver, kidney, and testicular cancer, as well as hypertension and low birth weight in children. Additionally, PFOA is persistent in the environment and is transported globally, as it is enriched in sea spray aerosol (SSA) particles over the Arctic Ocean. We aim to characterize the surface activity of PFOA at SSA proxy surfaces to better understand the pollutant enrichment in SSA particles. Surface tensiometry and infrared reflection-absorption spectroscopy are used to determine the surface adsorption constants via fitting to the Langmuir-Szyswkoski equation. PFOA exhibits enhanced surface activity in the presence of seawater cations in the low concentration regime, and decreased surface activity in the high PFOA concentration regime. It is speculated that the critical micelle concentration of PFOA is changing in response to the addition of seawater cations. Further study is underway to characterize the interfacial packing structure of the surfactant monolayer and the effect that the seawater cations have on the 2-dimensional arrangement of the PFOA molecules at the air-water interface. Future studies involve determining the impacts of dissolved organic compounds on the interfacial activity of PFOA at SSA proxy surfaces.

ACKNOWLEDGEMENTS

I would like to thank Kimberly Carter-Fenk for her guidance and support throughout this project and my research career, as well as her assistance in designing the experiments and analyzing the data. I would also like to thank the Allen Lab for providing support and ideas for this project and Dr. Heather Allen for providing me with the opportunity to pursue my research interests and for encouraging me to continue to seek out a career in research. Lastly, I would like to thank the College of Public Health, the College of Arts and Sciences and the Department of Chemistry and Biochemistry for the financial support of this project.

PUBLICATIONS

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Chapter 1: Background and Introduction

1.1 Sea Spray Aerosols

The sea-surface microlayer (SSML) is an important interface to study biological, chemical, and physical phenomena as it relates to oceanic life, stability, and climate parameters (Figure 1). The biological and chemical makeup of the SSML is diverse and relatively understudied; however, it is known that bacterioneustons and phytoneustons are the most abundant organisms at the SSML.[1] Moreover, the SSML is enriched with organic matter derived from marine microbiota in the water column.[2] Surface active organic matter can partition to the air-water interface via bubble entrainment. The SSML has been described as a “gel,” with a diverse range of particles, including microorganisms, organic matter, protein-like polymers, transparent exopolymeric particles, and polysaccharides.[1] Chemicals and biomolecules that are present in the SSML are then transported into the atmosphere through the production of sea spray aerosols (SSA).

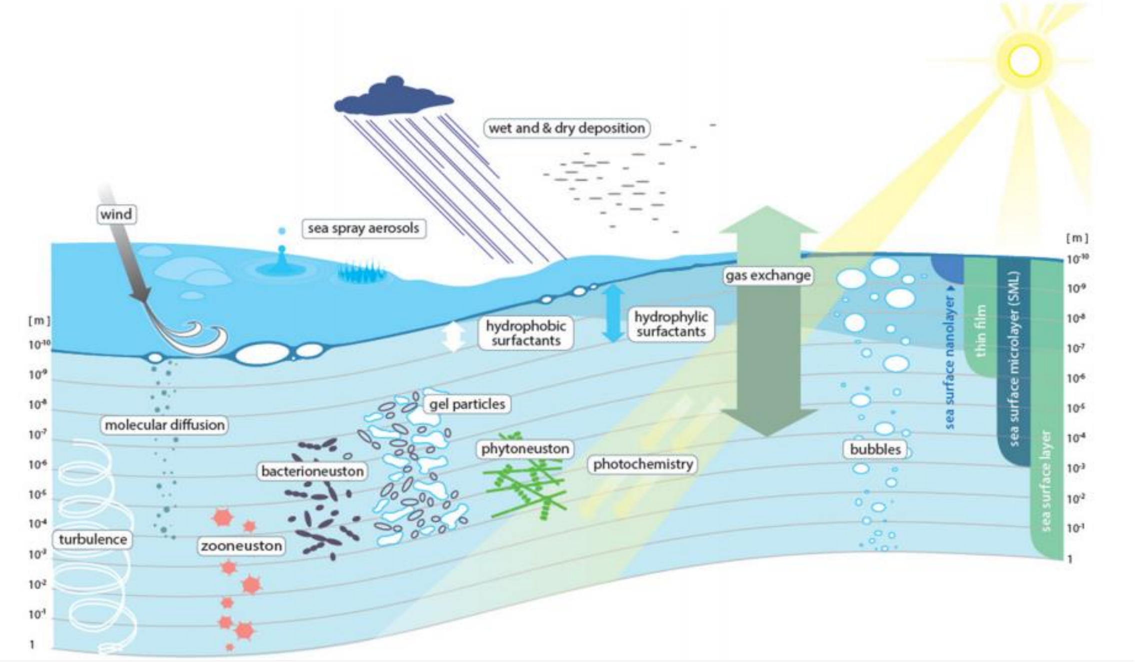


Figure 1: Characterization of the sea surface microlayer chemical composition and physical properties are imperative to understanding the production mechanisms, composition, and climate impacts of sea spray aerosol. (Reproduced from reference 1 - Published by Frontiers in Marine Science).

The chemical composition of SSA is driven by both the oceanic biota and the aerosol production mechanism at the SSML. SSA are formed through a process known as wave-breaking, wherein a wave crashes into the SSML and releases small aerosol particles, either film drops or jet drops, into the atmosphere (Figure 2).[3] A jet drop is formed during the bursting of a bubble at the surface, and when the water jet collapse and fragments, film drops are created.[4,5] The chemical and biological composition of SSA is highly heterogenous and the impact of ocean biology on the chemical composition of SSA is still an ongoing area of research.

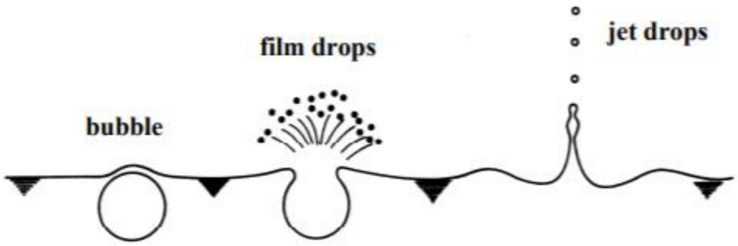


Figure 2: Schematic of wave-breaking that leads to bubble bursting and the creation of SSA. (Reproduced from Reference 6 - Published by the European Geosciences Union).

1.2 Surfactant Enrichment in Sea Spray Aerosols

At the SSML, there is a preferential enrichment of surfactants driven by the intermolecular interactions of amphiphilic molecules at the air-water interface.[6] When SSA are formed from bubbles that become entrained in the water column and rise into the SSML, the SSA particles are enriched with surfactants and other molecules found at the air-sea interface.[6] The enrichment of surfactants in SSA are associated with the size of the aerosol particle, of which smaller aerosol particles experience higher amounts of enrichment.[7] While SSA is mainly composed of organic matter derived from marine biological and chemical processes, anthropogenic chemicals, such as linear alkyl sulfates used in cleaning processes, are found to be present and enriched in SSA.[8] These anthropogenic pollutants enter the ocean via water runoff.

1.3 Perfluoroalkyl substances as surfactants

1.3.1 Environmental persistence

Perfluoroalkyl substances (PFAS) have been used widely throughout consumer products and industries across the world for decades. The United States Environmental Protection Agency (EPA) launched the Perfluorooctanoic acid (PFOA) Stewardship Program in 2006 to reduce the amount of PFOA environmental emissions by 95% in the year 2015 due to concerns of the chemicals affect on human health.[9] Consequently, perfluorooctanoic acid (PFOA) was phased out of some industrial production in the early 2000's; however, it is still widely used in several industrial and commercial processes, such as in flame-retardants for aviation and in some photography and film products.[10] Some derivatives of PFAS can also degrade into PFOA and perfluorooctanesulfonic acid (PFOS), showing that the continued use of any PFAS compound in industrial processes might still pose a significant risk to the health of those exposed.[11] Moreover, a study from 2016 found that water supplies for over 6 million American residents were contaminated with PFOA and PFOS at a concentration that exceeded the EPA's health advisory limit of 70 parts per trillion, thus showing the persistent health risks caused by the continued use of these compounds.[10] PFOA and PFOS are highly resilient compounds that do not degrade naturally in the environment; thus even though production of these chemicals has declined or halted altogether, their presence is still concentrated in the areas that it was produced or dumped as waste.[12,13] PFAS also are able to bioaccumulate in organisms through a variety of mechanisms, including partitioning to proteins in the blood plasma, and the environmental persistence of the chemicals allow for them to be magnified at different tropic levels within an aquatic ecosystem.[14] Due to the environmental persistence of PFAS, the EPA created the PFOA Stewardship Program, the Stockholm Convention classified PFOA and PFOS as persistent organic pollutants, and Canada has worked to eliminate the production of these chemicals in the country.[15]

1.3.2 Physical and Chemical Properties

The chemical structure of PFAS was at first important to industries because of its hydrophobic and oleophobic properties, allowing the chemical to repel both water and oil. These properties made the chemical ideal for commercial and consumer products, such as non-stick cookware. However, the strength of the carbon-fluorine bond, with a bond enthalpy of 485 kJ/mol, which is approximately 25 kcal/mol stronger than the C-C bond, created a heat stable compound, allowing PFAS to persist in the environment.[16] Many PFAS are also resistant to acids, bases, and reductants due to their physical properties, which further contributes to the chemicals ability to persist in the environment, as well as demonstrating the useful nature of using PFAS in a variety of industrial processes. Moreover, these compounds can create aqueous film-forming foams, indicating the high surface activity of these compounds in aqueous solutions due to their aggregation at the air-water interface, creating foams and aerosolizing above the surface.[16]

1.4 Global Transport of Perfluoroalkyl substances via Sea Spray Aerosols

In the 2000's, PFAS was found in both the geographic area of production and within remote Arctic ecosystems.[17] PFAS, most notably PFOA, was detected in water samples and tropospheric aerosols over the Greenland Sea, East Atlantic Ocean, and the Southern Ocean.[18] There have been attempts to quantify PFAS concentrations in the troposphere in various locations; however, the PFAS concentrations are often below the instrumental limits of detection.[17] PFAS have been measured in the blood plasma of polar bears in Norway at higher levels than detected in human blood samples, indicating the global spread and environmental ubiquity of PFAS.[19] While PFAS can spread to different geographic areas in water and in the tissue of aquatic animals, the widespread distribution of these chemicals has led to several hypotheses of additional routes of transmission. In proxy sea spray aerosol experiments, it was found that PFAS are significantly enriched in sea-spray aerosol particles, where PFOA has enrichment factors greater than 10,000 when the SSA particles are smaller than 2.45 μm (Figure

3).[20] Moreover, a global simulation using the Norwegian Earth Simulation Model predicted that sea spray aerosols are partially responsible for the long-range contamination of PFAS across the world, as well as for reintroducing PFAS into terrestrial environments, mostly near coastal areas (Figure 4).[20] Understanding the mechanisms through which PFAS are enriched in the SSML and in SSA are imperative in determining their distribution through air, water, and biomass, as well as the risk of PFAS exposure to aquatic and coastal ecosystems and coastal communities.

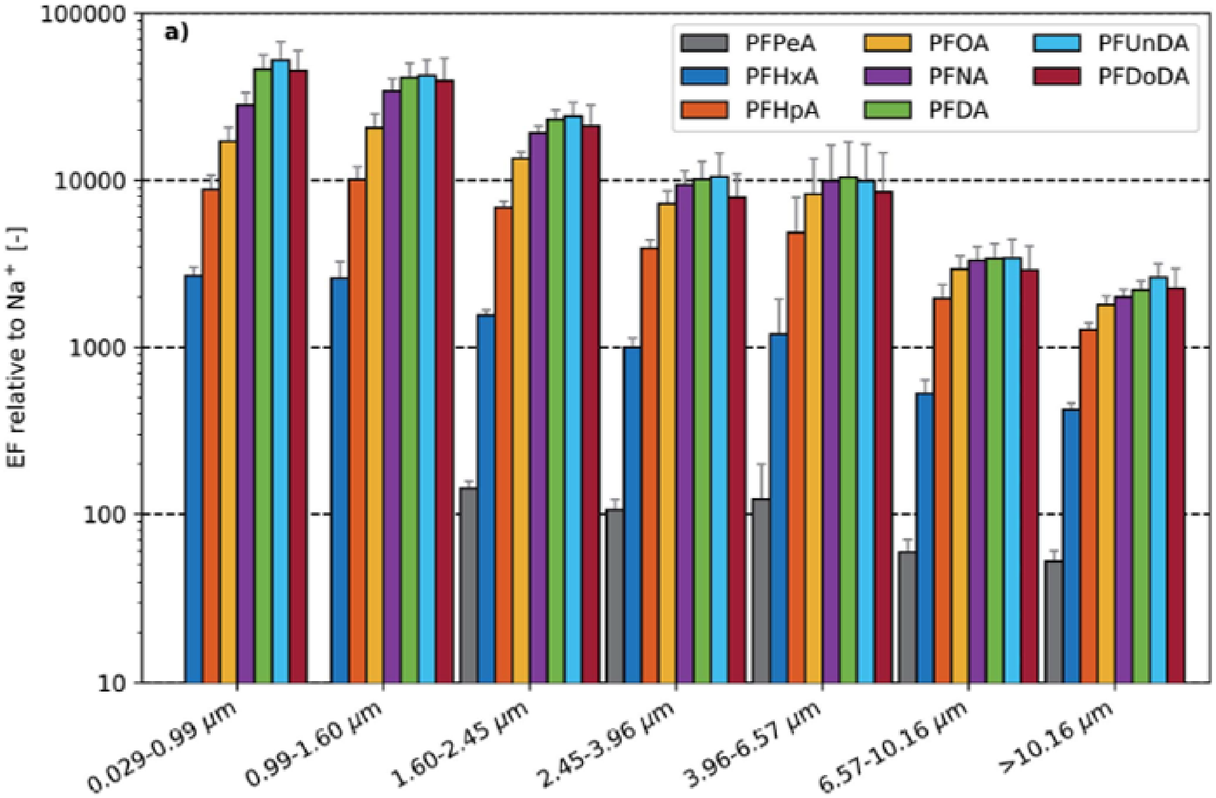


Figure 3: Enrichment factors of PFAS as a function of SSA particle size. (Reproduced from Reference 20 - Published by The Royal Society of Chemistry).

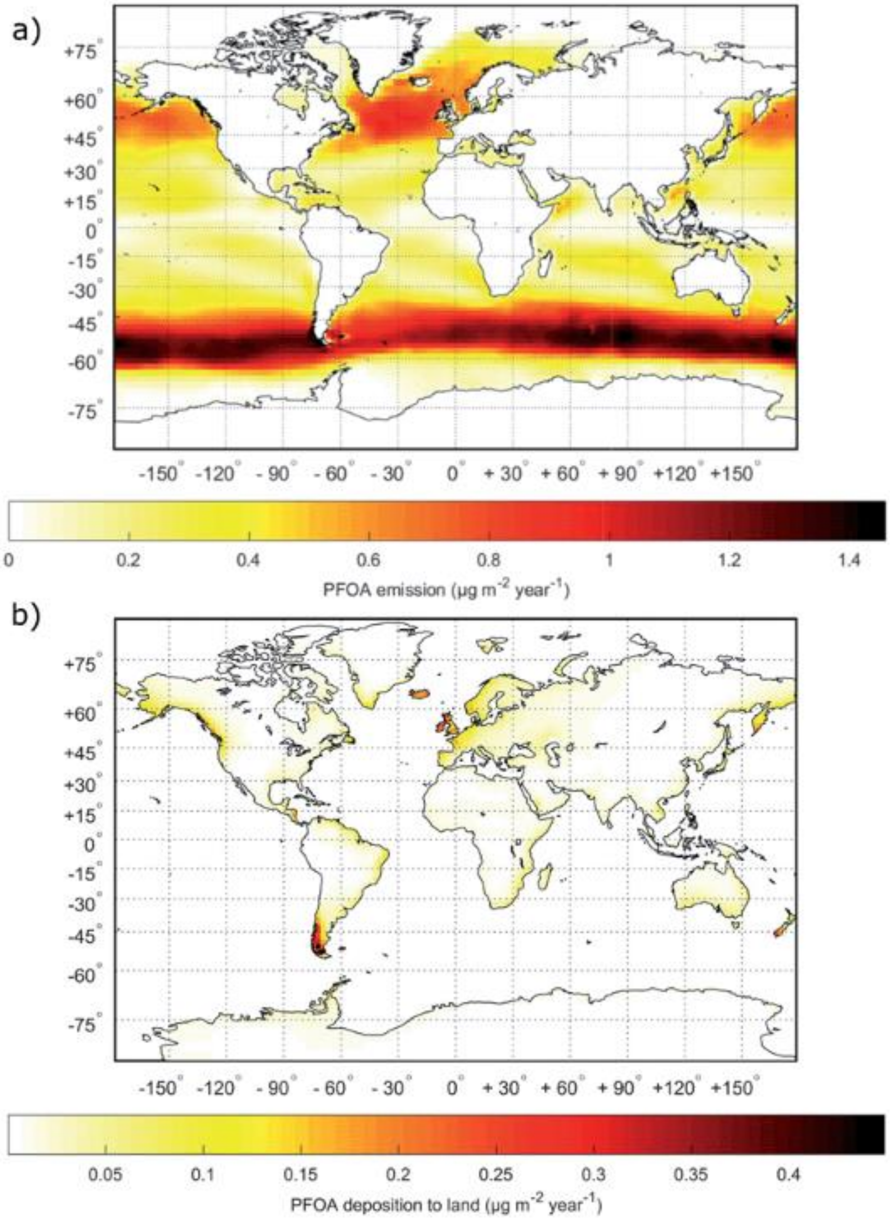


Figure 4: Modeling results of (a) PFOA emissions due to SSA and (b) PFOA deposition onto land masses. (Reproduced from Reference 20 - Published by The Royal Society of Chemistry).

1.5 Objectives and Specific Aims

The primary goal of this project is to determine the surface activity of PFOA through surface-sensitive techniques. Understanding the enrichment of this surfactant at the air-water interface is imperative in obtaining foundational knowledge about pollutant enrichment at the SSML and in SSA. Utilizing a surface-sensitive spectroscopic method, such as Infrared Reflection-Absorption Spectroscopy (IRRAS), the surface adsorption of PFOA can be characterized in order to collect a foundational understanding of the mechanism of enrichment of PFOA at the SSML. Surface tension experiments will be conducted in tandem with the IRRAS experiments in order to determine the surface activity of PFOA. The samples tested will include Na^+ and Ca^{2+} at ocean relevant concentrations, as well as a Neomarine solution to mimic seawater to determine if the cumulative interactions of molecules in seawater, instead of particular ions, affects the surface adsorption of PFOA and PFOS.

Chapter 2: Experimental Design

2.1 Solution Preparation

Perfluorooctanoic acid (PFOA, 96%, ACROS Organics™, Fair Lawn, NJ, USA; Figure 5) was used as received to create a stock solution of 2 mM PFOA in ultrapure water with a resistivity of 18.2 MΩ•cm (Milli-Q Advantage A10, EMD Millipore, Billerica, MA, USA). The stock solution was then diluted to 1 mM, 200 µM, 20µM, and 2µM PFOA in ultrapure water or in aqueous saltwater solutions. NaCl (Sodium chloride, 99.5%, for biochemistry, ACROS Organics™, Fair Lawn, NJ, USA) was baked at 650° C in a furnace (Fisher Scientific Isotemp® Muffle Furnace, Dubuque, IA, USA) for at least 10 h to remove residual organic impurities. The baked NaCl was dissolved in ultrapure water to prepare a 0.47 M NaCl subphase, or it was dissolved in the PFOA aqueous solutions. $CaCl_2$ (Calcium Chloride Dihydrate, Certified ACS, Fisher Chemical, Fair Lawn, NJ, USA) was used as received to prepare a 10 mM $CaCl_2$ solution in ultrapure water and in PFOA aqueous solutions. The pH of all NaCl, $CaCl_2$, and aqueous solutions were measured at 5.6 ± 0.2 due to acidification by atmospheric CO_2. Lastly, model seawater was prepared with NeoMarine (Brightwell Aquatics, Fort Payne, AL, USA) by adding 134.5g of NeoMarine reef salt per 3.785 L of ultrapure water (specific gravity of ~1.025 g/cm^3, pH of ~8.30, and alkalinity of ~7.5 dKH). The NeoMarine solutions were filtered using 0.22 µm PTFE membrane syringe filters (Fisherbrand™, Fair Lawn, NJ, USA) to remove insoluble particulates. PFOA aliquots were added to the filtered Neomarine solutions to create 2 mM, 1 mM, 200 µM, 20µM, and 2µM PFOA solutions in model seawater.

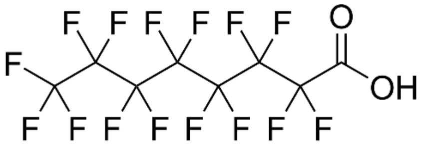


Figure 5: Chemical structure of perfluorooctanoic acid (PFOA).

2.2 Surface Tension Measurements

Surface tension experiments were conducted using a force tensiometer (Sigma 703D, Biolin Scientific, Espoo, Finland) and the Wilhelmy plate method (Figure 6). A platinum Wilhelmy plate was thoroughly cleaned with reagent alcohol (Histological Grade, Fisher Scientific, Fair Lawn, NJ, USA) and ultrapure water, and the plate was fired with a Bunsen burner until red hot. Aqueous solution aliquots (10 mL) were poured into acid-cleaned borosilicate glass petri dishes, and the dishes were rinsed with ultrapure water and reagent alcohol between measurements using different solutions. The surface tension of water was measured prior to each set of surface tension measurements to ensure that the calibration of the instrument and the height of the subphase was correct. The Wilhelmy plate was wetted in the subphase and then lifted completely out of the aqueous solution and tared. Lastly the plate was lowered until it made contact with the subphase surface, and the plate height was maintained for all surface tension measurements with that subphase. PFOA aqueous solution surfaces were allowed to equilibrate for 2 minutes prior to recording the surface tension, and all measurements were repeated at least 5 times.

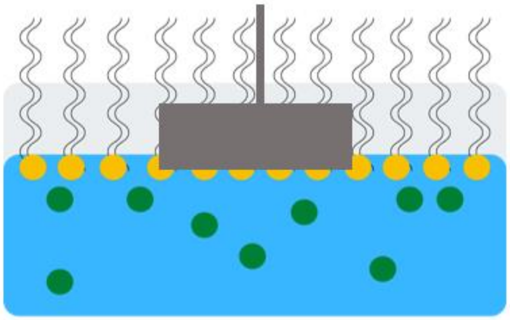


Figure 6: Schematic of the Wilhelmy plate method employed in the surface tension experiments.

2.3 Infrared Reflection Absorption Spectroscopy

Infrared-Reflection Absorption Spectroscopy (IRRAS; Figure 7) was used to characterize the surface activity of PFOA through a surface-sensitive spectroscopic method. To collect the IRRAS spectra, a Fourier transform infrared (FTIR) spectrometer (Spectrum 100, PerkinElmer, Waltham, MA, USA) with a liquid nitrogen-cooled HgCdTe (MCT) detector was used. The incident beam direction was modified by a planar, 2 inch gold mirror mounted to a breadboard inside the spectrometer with a 46° angle of incidence (relative to surface normal). The reflected light from the aqueous surface was then redirected toward the detector with a second gold mirror. The spectra were collected with unpolarized light in the single-beam mode as an average of 400 scans, and energy values were recorded every 0.5 cm^{-1} between 450 and 4000 cm^{-1}. Spectra with a 4 cm^{-1} resolution were plotted as reflectance-absorbance,

$$RA = -\log\left(\frac{R_m}{R_0}\right) \tag{2.1}$$

in which R_m is the reflectivity of the PFOA monolayer and R_0 is the reflectivity of the aqueous subphase without PFOA. Each experiment was repeated in triplicate and analyzed using Origin-Pro (OriginLab 9, Northhampton, MA, USA). For analysis of the C-F vibrational modes, the baseline was subtracted from each spectrum by fitting a line between endpoints 1118 and 1266.5 cm^{-1}. The baseline-subtracted spectra were averaged, and the error was plotted as one standard deviation (depicted as shading around the averaged spectrum).

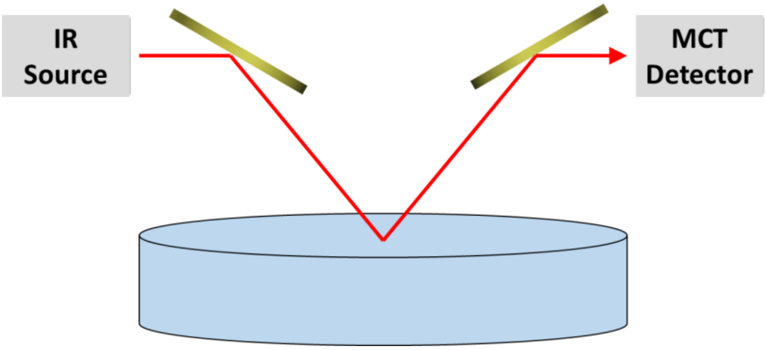


Figure 7: Schematic of the infrared reflection-absorption spectroscopy (IRRAS) experiments.

Chapter 3: Results and Discussions

3.1 Surface Tensions Measurements of PFOA Adsorption

The principle goal of this project was to quantify the surface activity of PFOA at sea spray aerosol (SSA) proxy surfaces and to determine the effect of different seawater ions on the interfacial adsorption of PFOA. Surface tension measurements were conducted to quantify the surface activity of PFOA at the aqueous interface, and the surface tension data for each subphase were fit to the Szyszkowski equation (Figure 8):

$$\gamma = \gamma_0 \left[1 - a \times ln\left(\frac{C}{b} + 1\right)\right] \tag{3.1}$$

in which γ is the surface tension of the PFOA solution, γ_0 is the surface tension of the aqueous subphase, C is the concentration of PFOA in bulk solution, a is a fitted parameter, and b is a fitted parameter corresponding to the PFOA surface activity. At low PFOA concentrations, the largest surface tension depression is observed in the Neomarine solution, showing that the complex mixture of salts in the Neomarine subphase has a significant affinity for the PFOA carboxylate headgroup. Consequently, the salts enhance the surface activity of PFOA in the low concentration regime. However, the 2 mM PFOA solution in 0.47 M NaCl exhibits the lowest surface tension, suggesting that Na^+ at a seawater-relevant concentration leads to the largest PFOA interfacial concentration.

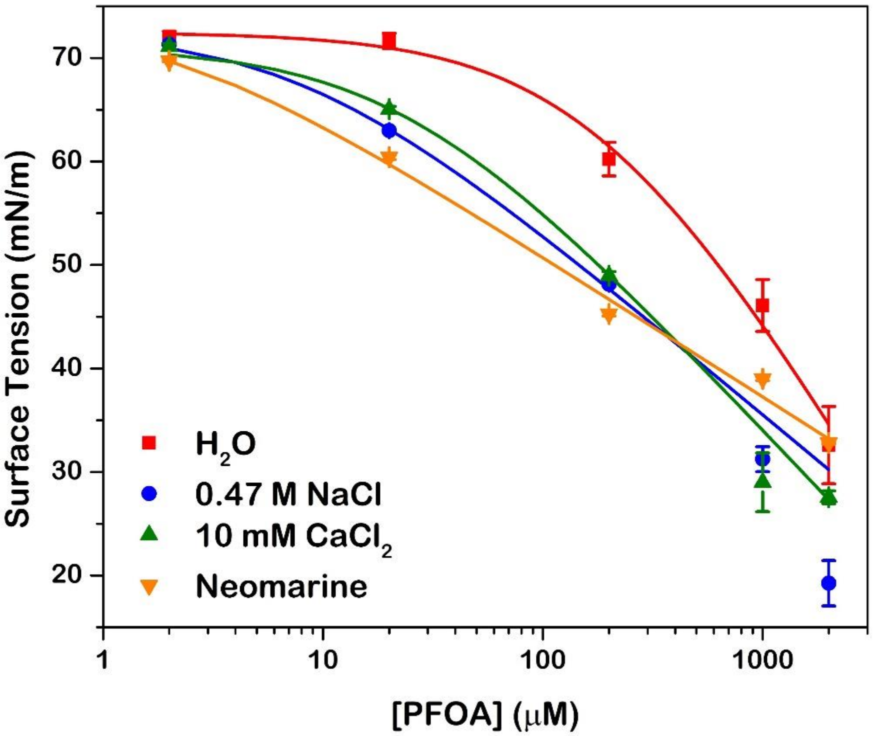
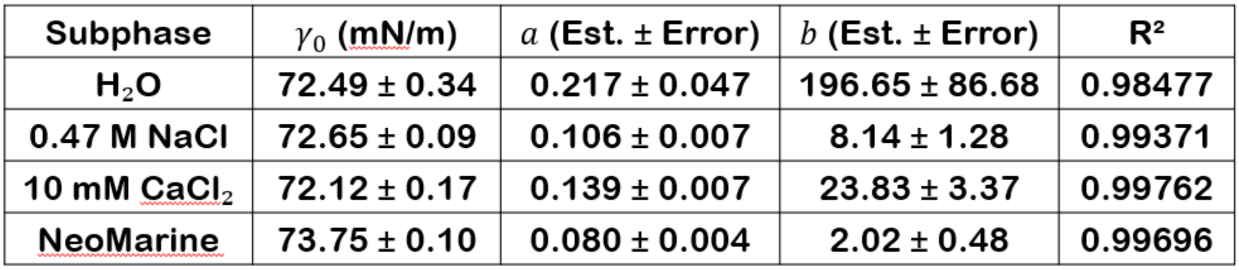


Figure 8: Surface tensions values of PFOA aqueous solutions fit to the Szyszkowski equation.

A quantitative analysis of PFOA surface activity can be conducted upon extracting the parameters from the Szyszkowski equation fits. A high surface activity of the surfactant shows that the PFOA molecules are enriched at the subphase interface, which is described by the fitted parameter *b* (Table 1). The surface activity of PFOA is the highest in the solutions with PFOA dissolved in H_2O, followed by the $CaCl_2$, NaCl, and Neomarine solutions. The surface activity of PFOA and H_2O is nearly two-fold higher than that of the $CaCl_2$ solutions with an R^2 value of 0.98, indicating a good fit of PFOA surface adsorption to the Szyszkowski equation.

Table 1: Values for the Langmuir-Szyszkowski Equation Fitted Parameters.



Subphase	γ_0 (mN/m)	a (Est. ± Error)	b (Est. ± Error)	R²
H_2O	72.49 ± 0.34	0.217 ± 0.047	196.65 ± 86.68	0.98477
0.47 M NaCl	72.65 ± 0.09	0.106 ± 0.007	8.14 ± 1.28	0.99371
10 mM $CaCl_2$	72.12 ± 0.17	0.139 ± 0.007	23.83 ± 3.37	0.99762
NeoMarine	73.75 ± 0.10	0.080 ± 0.004	2.02 ± 0.48	0.99696

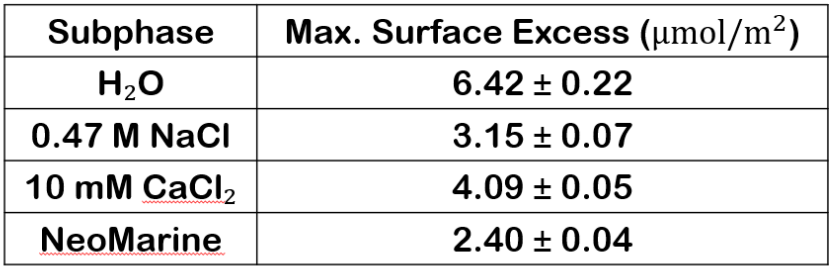
The Langmuir-Szyszkowski equation was then derived to determine the maximum surface excess Γ_{max} of PFOA on each subphase:

$$\Gamma = \frac{\gamma_0 a}{RT}\frac{C}{C+b}$$

$$\Gamma_{max} = \frac{\gamma_0 a}{RT} \tag{3.2}$$

where Γ is the surface excess as a function of surfactant concentration C, R is the gas constant, and T is the temperature. Surface excess is greater than 0 when the solute of interest is enriched at the interface, meaning that the solute is present in a larger concentration at the interface in comparison to the bulk. The PFOA and H_2O subphase has the highest Γ_{max} value, followed by the $CaCl_2$, NaCl, and Neomarine solutions; and PFOA is surface active on all subphase solutions (Table 2). This is the opposite trend observed by Costanza et al. in which the PFOA Γ_{max} value increased with increasing total dissolved solids (TDS).[21] The origin of this discrepancy is found in the fitted parameter a, where our values decrease with increasing salt concentration. The a values calculated by Costanza et al. increase with the aqueous salt concentration, indicating that further investigation into the Szyszkowski equation fitting is required.

Table 2: Values for the Langmuir-Szyszkowski Surface Excess Calculations.



Subphase	Max. Surface Excess ($\mu mol/m^2$)
H_2O	6.42 ± 0.22
0.47 M NaCl	3.15 ± 0.07
10 mM $CaCl_2$	4.09 ± 0.05
NeoMarine	2.40 ± 0.04

3.2 IRRAS of PFOA Adsorption at Proxy SSA Surfaces

While surface tension measurements yield a quantitative analysis of PFOA adsorption to proxy SSA surfaces, surface tension does not provide any molecule-specific information. Infrared reflection-absorption spectroscopy (IRRAS) allows for the collection of surface-sensitive infrared spectra by resonantly enhancing the vibrational modes of the chemical bonds of compounds on the surface of the aqueous subphase with infrared radiation.[22] This provides a molecular fingerprint of the proxy SSA surface because the bonds absorb different wavelengths of infrared radiation depending upon their unique chemical environments. The chemical bonds of interest are the C-F bonds which have three distinctive peaks: a CF_2 symmetric stretch at 1150 cm^{-1}, a CF_2 asymmetric stretch at 1210 cm^{-1}, and a CF_3 asymmetric stretch at 1245 cm^{-1}.[23] The IRRAS experiments used the same PFOA concentration range as studied in the surface tension experiments (Section 3.1). Moreover, solutions with different oceanic-relevant metal cations were also used to determine if the presence of these cations affected the surfactant activity of PFOA at proxy SSA surfaces.

Figure 9 shows the IRRAS spectra of PFOA on H_2O. The magnitude of the intensity of the CF_2 asymmetric, CF_2 symmetric, and CF_3 asymmetric stretch are the greatest for the high concentration regime of PFOA on H_2O. However, the C-F peaks cannot be resolved for the 2 µM, 20 µM and 200 µM PFOA solutions**.** Figure 10 is the IRRAS spectra of PFOA on 0.47 M NaCl. The C-F peaks are not as intense as that of PFOA and H_2O; however, it is important to note that the C-F peaks are resolvable for all concentrations tested, indicating that NaCl increases the PFOA surface activity at low PFOA concentrations. Figure 11 shows the IRRAS spectra of PFOA on 10mM $CaCl_2$. Here, all PFOA concentrations are resolvable, although the 2 µM PFOA sample has very low intensity. Figure 12 is the IRRAS spectra of PFOA on a Neomarine aqueous subphase. The 1 mM and 2 mM PFOA solutions have similar peak intensities for each of the C-F peaks, indicating that the PFOA solution is near the critical micelle concentration (CMC).

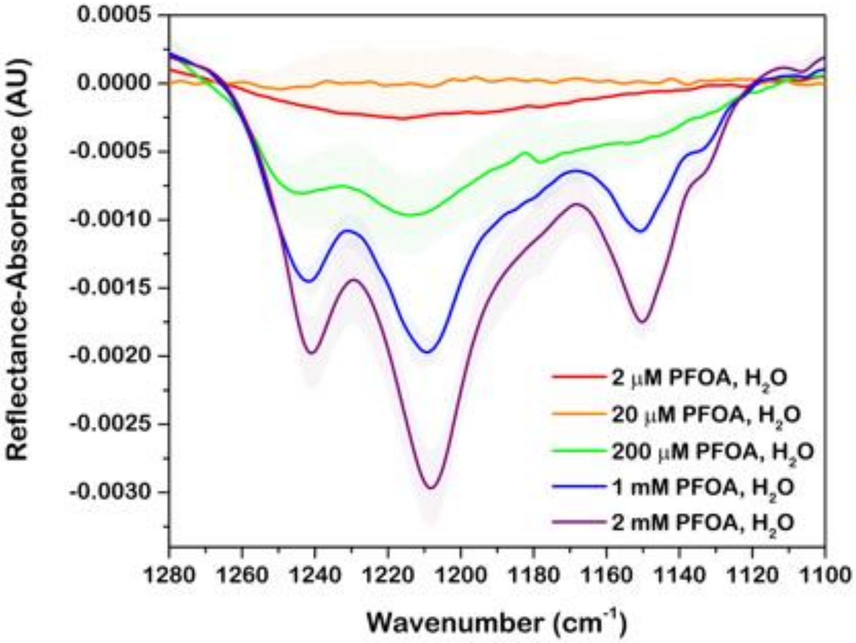


Figure 9: IRRAS spectra of PFOA on H_2O.

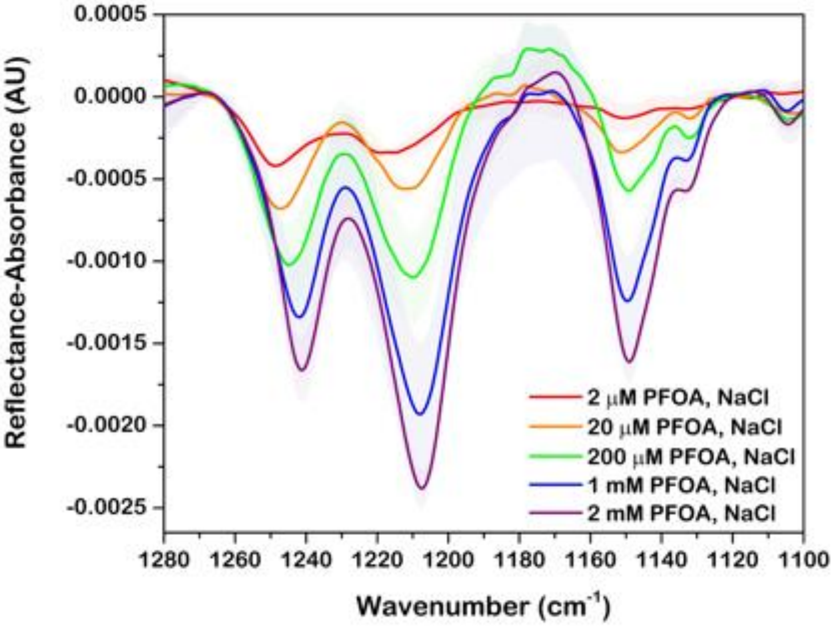


Figure 10: IRRAS spectra of PFOA on 0.47 M NaCl.

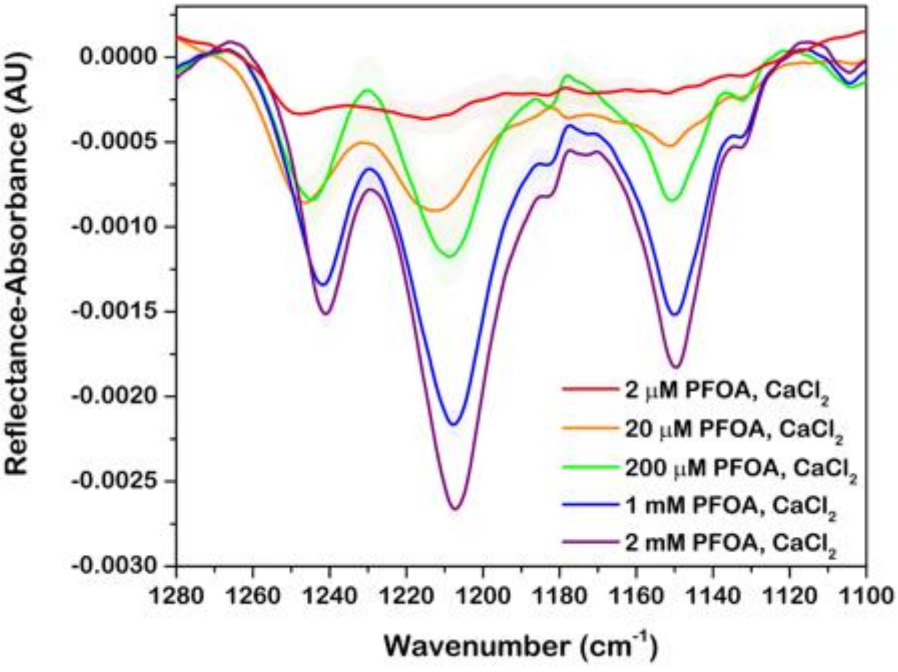


Figure 11: IRRAS Spectra of PFOA on 10 mM $CaCl_2$.

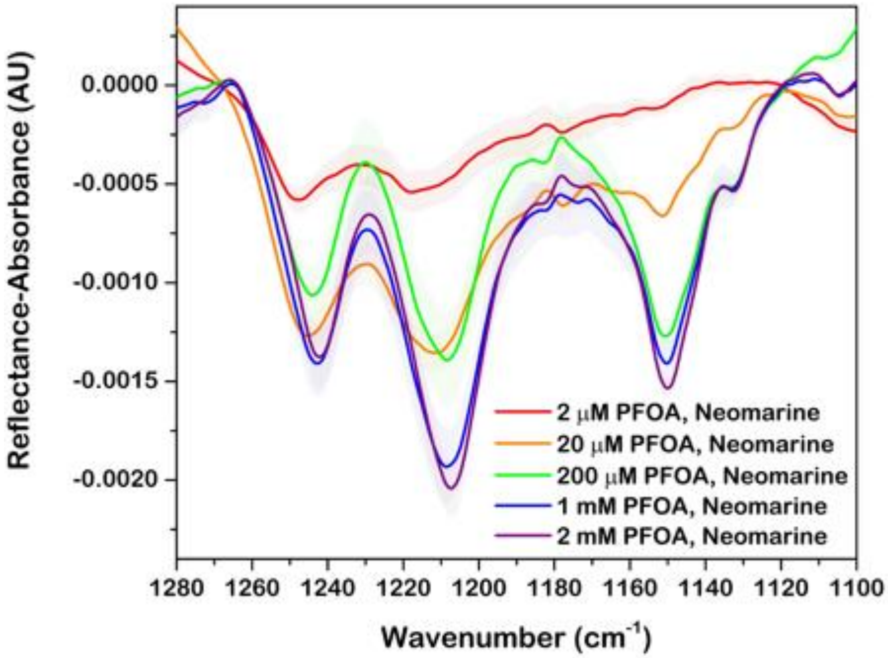


Figure 12: IRRAS Spectra of PFOA on the Neomarine aqueous subphase.

The IRRAS spectra of PFOA interfacial adsorption suggests that there is a change in thermodynamic surface activity and intermolecular interactions between the different aqueous subphases and the PFOA molecules. At each C-F peak, there is a shift in the center wavelength as a function of PFOA concentration. As the concentration increases, the wavenumber shifts towards a lower value, showing that there is a red-shift occurring at the air-water interface. Thus, as the concentration of PFOA increases, the intermolecular interactions between PFOA surfactants at the air-water interface increase due to the reduced interfacial area at high surface densities. Consequently, the C-F chemical bonds absorb infrared radiation at lower frequencies.

Figure 13 shows the shifts in wavenumber from the center wavelength of the CF_2 symmetric stretch. Because the 2 µM, 20 µM, and 200 µM PFOA solutions on H_2O are not resolvable for the CF_2 symmetric stretch, only the 1 mM and 2 mM PFOA spectra on H_2O are included in this analysis. The 200 µM PFOA spectrum on H_2O is resolvable for the CF_2 asymmetric and CF_3 asymmetric peaks, so those are later analyzed. Here, the PFOA on Neomarine aqueous subphase exhibited the largest wavenumber shift as a function of concentration from approximately 1154 cm^{-1} in the 2 µM PFOA spectrum to approximately 1150.5 cm^{-1} in the 2 mM PFOA spectrum. This suggests that the Neomarine aqueous subphase facilitated the greatest rearrangement of PFOA molecules at the air-water interface into a more favorable conformation.

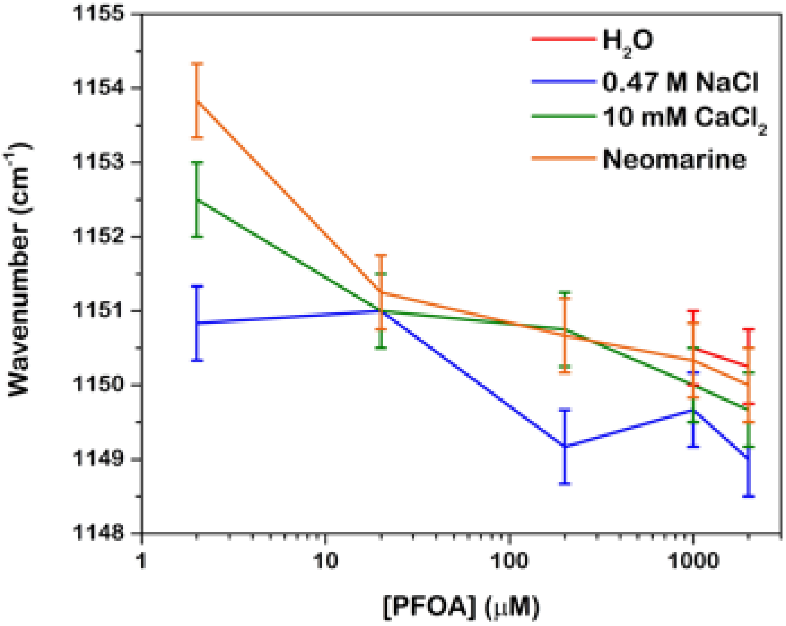


Figure 13: Graph of the wavenumber shift in the CF_2 symmetric peak.

The wavenumber shifts of the PFOA CF_2 asymmetric peak are found in Figure 14, where the 200 µM, 1 mM and 2 mM PFOA spectra on H_2O deviate significantly from the saltwater subphases. While the lower concentrations in this series cannot be analyzed, PFOA on water exhibits a strong change in intermolecular packing at the air-water interface between 1 mM and 2 mM PFOA. The center wavelength shifts of the PFOA CF_2 asymmetric peak on saltwater subphases exhibit similar trends, with the Neomarine subphase causing the largest red-shift. Figure 15 shows the wavenumber shifts in the CF_3 asymmetric stretch. The wavenumber shifts between 200 µM and 2 mM PFOA are large in magnitude and approximately the same for the H_2O, NaCl, and $CaCl_2$ subphases. The Neomarine subphase exhibits the smallest magnitude red-shift in this concentration regime. This is possibly because the Neomarine salts induce PFOA reorganization at the air-water interface at lower surfactant concentrations due to the increased surface activity.

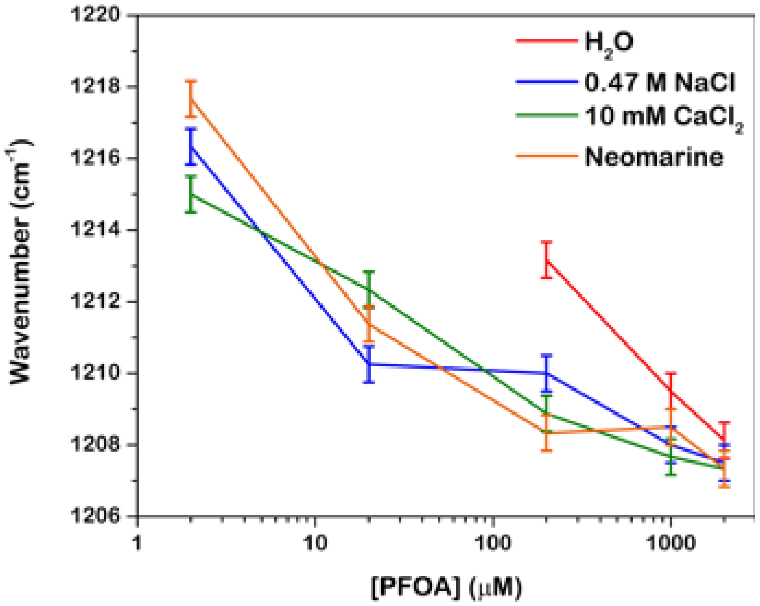


Figure 14: Graph of the wavenumber shift in the CF_2 asymmetric peak.

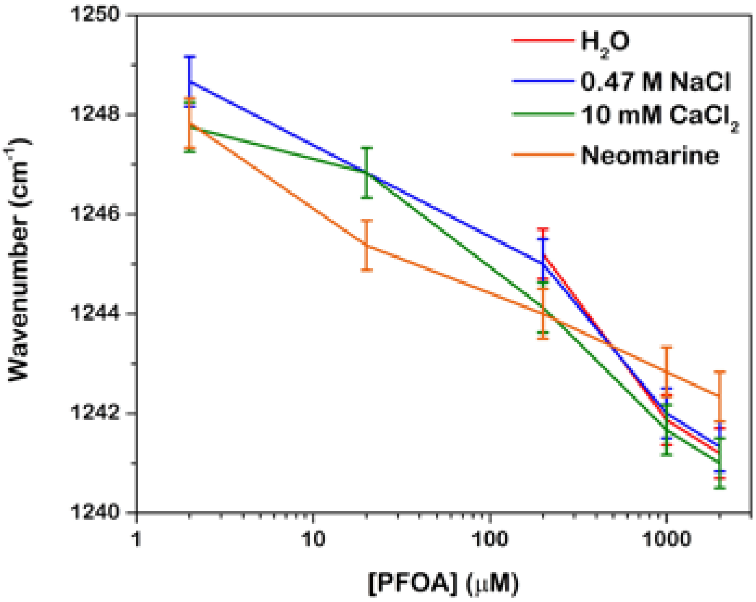


Figure 15: Graph of the wavenumber shift in the CF_3 asymmetric peak.

It is evident that the higher concentration regimes, 200 µM – 2 mM PFOA, lead to the greatest reorganization of PFOA molecules at the air-water interface, as shown in the -CF_3 vibrational modes of the PFOA molecules. The CF_2 symmetric stretch and the CF_2 asymmetric stretch are the most sensitive to changes in interfacial organization in the lower concentration regimes, with the exception of the CF_2 asymmetric stretch of PFOA adsorbed on H_2O. Moreover, a full monolayer of PFOA molecules is likely forming at the air-water interface at the 2 mM concentration. The rearrangement of PFOA molecules as a function of bulk concentration on proxy sea spray aerosol surfaces shows that these molecules are highly surface active and that their interfacial packing on SSA varies based on the composition of the aqueous system. It is imperative to be able to define the interfacial characteristics of PFOA on aqueous subphases in order to better understand how the pollutant behaves in aqueous systems and can be taken up into SSA particles, thereby travelling to pollute other areas.

3.3 Langmuir- Szyszkowski Adsorption Modeling

To extract quantitative surface activity values from the IRRAS spectra, a Langmuir adsorption model is used to fit the integrated area of the IRRAS PFOA C-F peaks:

$$I = I_{max} \frac{C}{C+b} \tag{3.3}$$

in which I is the integrated area of the PFOA solution, I_{max} is the maximum integrated area of all PFOA solutions, C is the PFOA bulk concentration, and b is the fitted parameter corresponding to surface activity. Figure 16 shows the Langmuir equation fits to the IRRAS integrated C-F peak area for each subphase, and Table 3 summarizes the surface activity values of the Langmuir fits in comparison to the surface tension Szyszkowski equation surface activity values. The low R^2 values of the Langmuir equation fits indicate that the IRRAS integrated peak areas of the PFOA C-F peaks do not fit an ideal Langmuir adsorption isotherm model. Hence, the IRRAS spectra are likely probing a different physical entity than the surface tension measurements. Qualitatively, the H_2O subphase exhibits the greatest PFOA surface activity, followed by NaCl, $CaCl_2$, and Neomarine aqueous subphases. Further experiments and modeling must be conducted to understand the differences between the two experimental techniques.

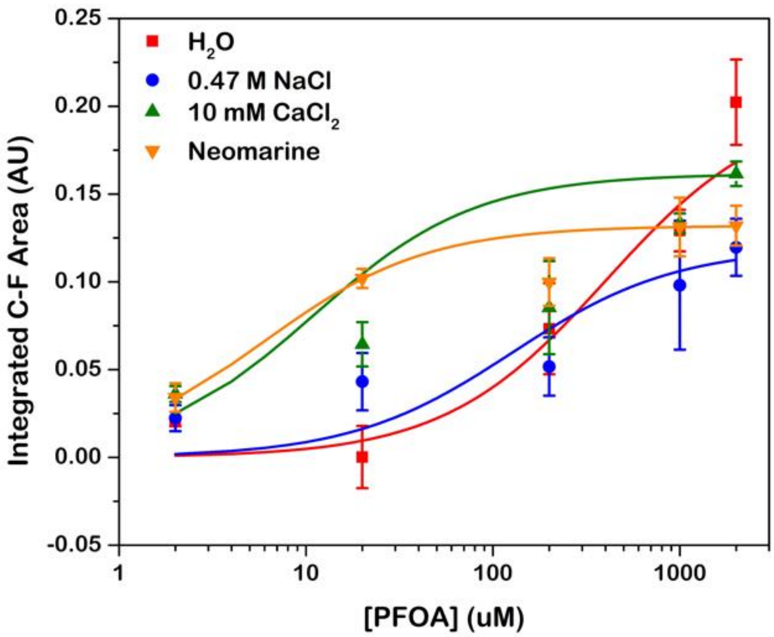


Figure 16: Integrated IRRAS C-F peak area of PFOA fitted to a Langmuir model.

Table 3: Surface Activity Values for IRRAS Langmuir Equation Fits and Surface Tension Szyszkowski Equation Fits.

	IRRAS Fitted Parameters		**Surface Tension Fitted Parameters**	
Subphase	**Surface Activity, *b***	**R^2**	**Surface Activity, *b***	**R^2**
H_2O	**405.0 ± 366.1**	**0.59655**	**196.65 ± 86.68**	**0.98477**
0.47 M NaCl	**127.3 ± 111.6**	**0.62244**	**8.14 ± 1.28**	**0.99371**
10 mM $CaCl_2$	**10.9 ± 6.6**	**0.85559**	**23.83 ± 3.37**	**0.99762**
Neomarine	**6.0 ± 1.2**	**0.93910**	**2.02 ± 0.48**	**0.99696**

Chapter 4: Public Health Implications

4.1 Overview of Health Effects

Perfluoroalkyl substances (PFAS) are ubiquitous in modern life; they are found in nonstick cookware, drinking water, upholstery, and house dust.[24] The United States National Health and Nutrition Examination Survey (NHANES) in 2011 and 2012 found that 97% of individuals had detectable amounts of perfluoroalkyl compounds in their serum.[25] Perfluorooctanesulfonic acid (PFOS) was phased out of industrial production in the United States in the early 2000's; however, it is still used in flame retardants, upholstery, and other consumer goods.[26] Exposure to PFAS represents risks for every person, including professional skiers through occupational exposure, and in children that are experiencing a decrease in serum vaccine antibody concentrations from exposure to PFAS.[27,28] Furthermore, perfluorooctanoic acid (PFOA) and PFOS are environmentally persistent molecules, deemed "forever chemicals," and their presence is still concentrated in water systems and soil where the chemicals were dumped as waste.[29] The half-lives of PFOA and PFOS in humans have been determined to be approximately 3.8 and 5.4 years, respectively.[30] Current measurements of PFOA and PFOS in human serum are indicative of long-term environmental exposure and long half-lives of these chemicals in humans. Thus, the physiological effects due to acute and chronic exposure need to be characterized in order to understand the cumulative adverse health effects from PFAS for children, adults, and those who are at risk of occupational exposure.

4.2 Perfluoroalkyl Substances Effects on Human Health

The accumulation of PFAS in human serum appears to vary based on a variety of factors that are not yet understood, such as gender. A study that evaluated human serum from Red Cross Blood Banks also found that men had a higher geometric mean of PFOA and PFOS in their serum; however the data showed that age did not have a significant impact on the concentration of PFOA or PFOS in their human serum samples.[31] It is unknown currently whether humans can metabolize larger perfluoroalkyl substances (PFAS) into PFOA or PFOS, and if so, if there are any toxic byproducts of such a reaction. However, the geometric average of PFOS in human serum samples was approximately 34.9 parts per billion, ranging from 4.1 parts per billion to 1656 parts per billion.[31] The occupational hazard of PFAS can be seen in the abnormally high concentrations of PFOA and PFOS in the blood samples of those that work in factories that use these chemicals, and those that live near the factories.

Moreover, a study that analyzed NHANES data from 1999-2000, 2003-2004, and 2005-2006 found that there is an association between thyroid disease in women and their serum levels of PFOA, most notably with women that were being treated for thyroid disease at the time of the sample.[32] While there are several animal studies that show a link between exposure to PFAS and the disruption of thyroid hormones, the epidemiological human studies show various outcomes of the association between PFAS exposure and thyroid hormones. There is evidence that PFOS can act as a competitor to thyroxine by competitively binding to the human thyroid hormone transport protein transthyretin.[33] This type of competitive inhibition could be an explanation for the abnormal thyroid hormone levels seen in animal studies and can also be used to explain the ability of PFAS to bioaccumulate in humans.

One of the target organs for toxicity of PFOS is the liver. The liver has been shown to be a large source of PFOS bioaccumulation, where PFOS has altered the ability of the liver cells to metabolize lipids which can lead to oxidative stress and can impact the cell cycle.[34] PFOS can also cause neurotoxicity by altering calcium ion channels, causing neuroinflammation, and altering physical characteristics of synapses, such as plasticity and shape.[34] PFOA has been found to induce weight gain in rats in a dose-dependent manner, to affect the morphology of the kidney, and to cause discoloration and increases in mass of the liver.[35] Furthermore, cancer risks for humans have been studied in accordance with the C8 Health Project, where a large scale epidemiological study concluded that there was an association between those that were chronically exposed to high concentrations of PFAS, such as those living near a facility that creates or uses the chemicals, and testicular and kidney cancer.[36]

There is a multitude of evidence to show that pregnant women and young children are especially at risk for adverse health effects if exposed to PFAS. The most common route of exposure to PFAS is ingestion of contaminated water, and there is also evidence showing that PFAS, specifically PFOA, can pass the placental barrier with little hindrance, thus exposing an unborn child to PFOA.[37] In early life, human serum levels of PFOA are found to be nearly 40% higher than PFOA serum levels in adults due to the ability of PFOA to cross the placenta, along with the combined exposure of contaminated breastmilk and water.[38] Additionally, PFOA levels in the serum of pregnant women were found to be directly proportional to BMI and a larger waist circumference.[39] Moreover, animal studies indicate that prenatal exposure to PFOS reduced the survival of mice in a dose-dependent manner; and those that survived had a low body weight, hypothyroxinemia, low levels of thyroxine, and neurodevelopmental effects.[40] PFAS exposure to neonates has also been shown to impact the development of the fetal liver by extensively altering genes related to lipid catabolism and overall lipid homeostasis.[41] The effects of PFAS on human health are transgenerational; neonates are exposed through placental barrier transfers, breast milk, and the water they drink. The additive exposure of several species of PFAS and their cumulative health effects need to be better characterized to understand the burden these chemicals have on human morbidity. Consequently, PFAS have been listed as persistent organic pollutants under the Stockholm Convention.

4.3 Routes of Exposure

Humans are exposed to PFAS through adsorption, ingestion and inhalation pathways. Because these chemicals are ubiquitous in the environment, they have contaminated numerous water sources all over the world. Perfluoroalkyl compounds have also been found in household dust and dryer lint, which disproportionately affects children because they not only inhale these particles, but they also ingest them through playing with dusty toys and on the floor.[42] PFAS can also travel throughout the troposphere and have been hypothesized to be able to remain in the troposphere for up to 20 days, thus showing how the chemicals can move long distances in a heterogenous fashion.[43]

While humans and other organisms can be exposed to PFAS at a large geographic distances from the contamination, adsorption and ingestion at the source of the contamination is also a common mechanism of exposure. The use of PFAS in the food industry has infiltrated individual homes through the use of Teflon cookware and the restaurant industry with non-stick technology and food packaging.[24] PFAS can contaminate many food products, especially animal products such as beef and fish products, where PFOS and PFOA are the dominant contaminants.[44] Fish, in particular, are a source of PFAS exposure for many humans and are expected to continue to remain so as water systems are polluted with PFAS which bioaccumulate in aquatic life.[44,45] A pharmacokinetic model was used to determine that exposure to PFAS in most of the population is through dietary intake; however, for those that live near a site contaminated with PFAS, ingestion of drinking water leads to the highest exposure rate of PFAS.[46]

A study from 2016 found that water supplies for over 6 million American residents were contaminated with PFOA and PFOS at a concentration that exceeded the EPA's health advisory limit of 70 parts per trillion, thus showing the health risks that are persisting due to the continued use of these compounds.[47] Here, studies have been conducted to determine the risk of exposure to PFASs, and PFOA or PFOS were found in blood samples from 97% of the individuals that were tested.[48] The C8 Health Project assessed the exposure of PFOA to residents that lived near a fluoropolymer facility in Ohio and West Virginia. Here, it was determined that the water systems became contaminated with PFOA and those that used a private well had a serum PFOA concentration that was 20% higher than the national average.[49] There was a 141.5 µg/L increase in the concentration of serum PFOA levels for every 1.0 µg/L increase of PFOA in the drinking water.[49] Ingestion of contaminated water continues to be a significant source of PFAS exposure; and while the EPA has a recommended limit of 70 parts per billion of PFAS in drinking water, this is not an enforceable or mandatory limit, so many Americans remain at risk of exposure.

4.4 Aerosolization of Perfluoroalkyl Substances Effect on Lung Function

Inhaling PFAS can have a variety of health effects, such as inducing asthma in those that are exposed at a young age, increasing airway inflammation, and affecting the overall function of the airway.[50] PFAS also affects the function of lung surfactants, the induction of chemokines and cytokines in an immune response, and lung macrophages, a phagocytic immune cell, through the inhalation of PFAS, as well as PFAS that are circulating in the body.[51,52] In an experiment that fluorescently labelled PFOS and introduced it into mice through food and injection, it was found that PFOS accumulated in lung tissue and in unborn mice pups; and early deaths of the pups was attributed to damage in lung development.[53] PFOA has also been found to have an effect on the lung development in fetal mice by altering the genes necessary for lung development in a dose-dependent manner during fetal development, most of which were involved in lipid homeostasis.[40] It has been hypothesized that PFOA and PFOS can affect the ability of alveolar type II cells to create pulmonary surfactant, thus leading to a variety of lung and respiratory issues.[35] A thorough understanding of the adverse health effects that can occur in human lungs due to inhalation of PFAS, as well as prenatal exposure, is imperative in creating a risk assessment for these chemicals. Moreover, because PFAS can be transported globally through sea spray aerosols (SSA) and can be deposited onto terrestrial coastlines, it is imperative to study these health effects and the additional burden that can be placed on humans and animals living near the coasts.

Chapter 5: Conclusions and Future Directions

5.1 Conclusion

Seawater cations have a significant effect on the interfacial surface adsorption of PFOA. Characterizing the surface activity of PFOA is imperative in understanding the enrichment of this surfactant in the sea surface microlayer (SSML) and in sea spray aerosols (SSA). In the presence of seawater cations, the low concentration regime of PFOA exhibits enhanced surface activity, whereas the high concentration regime of PFOA exhibits decreased surface activity. It is believed that the presence of the seawater cations is inducing a change in the critical micelle concentration of PFOA, thus in the higher concentrations on PFOA, the surfactant molecules are forming micelles and pulling away from the surface leading to the observed decrease in surface activity. This finding is important in understanding the pollutant nature of PFOA and its ability to be enriched at the SSML in low pollutant concentrations.

Since PFOA is enriched at the SSML and can be aerosolized, those that live in coastal communities are at risk of chronic exposure to PFOA due to the terrestrial dump of SSA. Moreover, it is possible that PFOA pollutants in other water systems can be aerosolized and travel to different geographic areas. This is a public health risk due to the plethora of adverse health effects that can be caused by chronic exposure to PFOA, as well as acute exposure. Moreover, as sea levels rise and runoff increases, it is important to understand how PFOA will be enriched or interact with other organic and biological matter at the air-water interface and the effects that can have on human health.

5.2 Future Directions

Perfluorooctanesulfonic acid (PFOS) is also a common perfluoroalkyl substance (PFAS) that has polluted waterways and has been shown to have adverse effects on human health. We aim to characterize the surface activity of PFOS through infrared reflection absorption spectroscopy (IRRAS) and surface tension through identical experimentation as was done with PFOA. The use of Brewster Angle Microscopy (BAM) will also be utilized in order to view the 2-dimensional packing structure of the PFOA and PFOS monolayer and how that interfacial packing structure changes in response to seawater cations. Moreover, we aim to determine the impacts of other dissolved organic compounds on the interfacial activity of PFOA and PFOS at SSA proxy surfaces through IRRAS and surface tension experiments.

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