Coating Polyurethane with Palmitoleic Acid and Bovine Serum Albumin to Prevent the Host Response to Foreign Materials

*Sheherbano Hussain, Zoha Babar, Jimmy Hadid, & Jacqueline McLaughlin**

Department of Biology, Penn State Lehigh Valley, Center Valley, PA

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Students: skh5406@psu.edu, zph5044@psu.edu, jjh5916@psu.edu,*
Mentor: jxm57@psu.edu

ABSTRACT
Macrophages are cells of the immune system that play a pivotal role in the host inflammatory response by attacking and engulfing any foreign molecule not seen as 'self.' They also help regulate the host response by releasing a variety of cytokines and growth factors that act as signals to other cells to amplify the host response. However, the host response causes degradation of implanted medical devices composed of polyurethane as well as other synthetic materials which it does not identify as self. Research was undertaken to investigate the potential of coating polyurethane with the self-like molecules palmitoleic acid and albumin to reduce or prevent the body's host response from damaging implanted medical devices. Using an *in vitro* THP-1 bioassay, polyurethane films coated with palmitoleic acid and bovine serum albumin showed a reduction in macrophage adherence. The individually coated palmitoleic acid and bovine serum albumin films significantly reduced the number of cells attached to the films with increasing concentration while the films coated with the conjugate of both showed no statistical difference. This suggests the potential role of self-like molecules in reducing the inflammatory response to foreign materials.

KEYWORDS
Macrophages; host inflammatory response; THP-1; prosthetics; palmitoleic acid; bovine serum albumin; cytokines; immune system

INTRODUCTION

Macrophages are large white blood cells that engulf and digest cellular debris, foreign substances, microbes, cancer cells, and anything else seen as foreign. Thus, they play a key role in the human body's 'host response,' which is the defense mechanism and reaction against the presence of foreign molecules in the body. It constitutes a variety of mechanisms that contribute to how the body responds to anything that is foreign or non-self. Macrophages are formed from monocytes, which are white blood cells that differentiate from stem cells produced in the bone marrow and reside in the extracellular matrices between cells in the body's tissues. To differentiate between foreign molecules and self, a number of phagocytic membrane receptors have evolved on the surface of macrophages that aid the immune system in the ability to recognize and respond to foreign materials and tolerate self-components of the body. These receptors can be identified using flow cytometry or immunohistochemical staining.[1]

Recognition of a foreign molecule by a macrophage through receptor binding involves phagocytosis. A pocket called a phagosome is formed around the unwanted material. Enzymes digest and degrade the unknown materials.[2] In addition, macrophages also work to help regulate the inflammatory response by producing and releasing a variety of cytokines and growth factors. For activation, cytokines and growth factors send signals to other cells to amplify the host response. On the other hand, anti-inflammatory cytokines like interleukin-10 can mediate the host response.[3]

One of the most important functions of macrophages is that they can activate the adaptive immune system. After a macrophage has phagocytized and digested foreign material, it displays some of the broken-down proteins (antigens) on its cell surface. These antigens act as identification signals for Helper T cells. Helper T cells can "read" these signals and tell what kind of particle the macrophage has ingested. If the T cell determines the macrophage has eaten something harmful (a pathogen), it can trigger a powerful reaction towards the specific pathogen.[4]

THP-1 cells are a human monocyte-derived macrophage cell line derived from the peripheral blood of a one-year old male infant with acute monocytic leukemia.[5] These cells are considered an ideal cell line for growing suspension cell cultures and monolayers on experimental surfaces. THP-1 cells have been used to study the host response to biomaterials and biocompatibility because they are an excellent model of monocytes to macrophage differentiation.[6, 7] This knowledge is essential in medicine, bioengineering, and biomaterial science, since the host response detects foreign materials through activated macrophage attack via

phagocytosis, release of proinflammatory cytokines, and neutrophil activation – ultimately degrading the material.[8] Device failures due to the host response can lead to harmful health effects. Moreover, the pathophysiology in accordance with this biological process following medical device implementation exhibits a considerable healthcare burden.[9]

Specific fatty acids have been shown to affect control processes in inflammatory responses and have been researched in this respect. Due to their hydrophobic nature, fatty acids are incorporated into cell membranes. Research shows that they can bind to membrane receptors or change intracellular protein activation, thereby altering cellular function.[10]

Palmitoleic acid (PA) is an n-7 monounsaturated fatty acid secreted by adipose tissue. Studies have shown that monounsaturated fatty acids, like palmitoleic acid, modulate the secretion of important transcription factors involved with inflammatory pathways by inhibiting NF-kB, which is essential for cytokine production.[11] This occurred specifically by a decrease in the Th1, Th17, and CD28 responses, and an increase in the inhibitory receptor CD95.

In cultured macrophages, evidence has also been shown that PA decreases the level of proinflammatory cytokine expression. Macrophages were isolated from the intraperitoneal cavity of mice and were exposed to PA conjugated with albumin for 24 hours in culture. RNA was isolated and gene expression was quantified to determine the relation to metabolic and inflammatory pathways. PA was observed to stimulate anti-inflammatory effects by inhibition of the inflammasome pathway, which constitutes the complex of proteins associated with activating the inflammatory response. PA decreased the expression of inflammatory markers, specifically NFκB and IL-1β.[12]

In the following experiment, to further research potential deterrents of the human host response to implanted biomaterials, PA was investigated for its ability to decrease the number of adhered THP-1 cells to polyurethane *in vitro*, a commonly used polymer in implantable devices. It was reasoned that there would be a significant decrease in THP-1 cell adherence to polyurethane films coated with PA through the THP-1 Attachment Assay.[13] Since using PA in a cell-based assay may prove to be challenging due to its hydrophobic nature, PA conjugated with albumin was also investigated to assist in the coating process. Human albumin is a blood protein that can serve as an adhesion location for macrophages and other cells of the immune response once an implant has interacted with the tissue. However, since bovine serum albumin (BSA) is commonly used in THP-1 cell culture protocols, it is a molecule both recognizable by THP-1 cells and unable to increase the host response.[14] Due to its negative charge, it can bind water, salts, fatty acids, vitamins and hormones and carries these bound components between tissues and cells.[13] With fatty acids in particular, BSA creates an aqueous-soluble reagent that can be absorbed and employed by cells as exemplified through its known conjugation with palmitate.[16] With this composite knowledge, the following hypothesis was developed and tested: there will be a significant decrease in activated THP-1 cell (macrophage) adherence to polyurethane films coated with the self-like molecules PA, PA conjugated to BSA, and BSA through the THP-1 Attachment Assay.[13]

METHODS AND PROCEDURES

A. Experimental Design for Cell Culture

The experimental design for this experiment is depicted in the schematic 24-well plate presented in Figure 1. Polyurethane control films coated with sodium chloride only, and experimental films coated with different concentrations of PA, PA conjugated with BSA, and BSA were prepared and utilized. For the first set of wells, the concentration of PA and BSA was 100 μM. As for the second set, the concentration of PA and BSA was 500 μM. Lastly, the third set of wells had films coated with 1000 μM of PA and BSA. The ratio for PA conjugated with BSA in each experimental concentration equaled 1 mM of PA to 0.17 mM of BSA (see below section, Coating of Polyurethane Films).

B. Coating of Polyurethane Films

Sheets of polyurethane films were obtained from Children's Hospital of Philadelphia (CHOP, PA), and cut into 1 cm x 0.5 cm rectangles, then placed into the wells of a 24-well culture plate - all in a vented hood using sterile scissors and forceps. A final concentration of 100 μM, 500 μM, and 1000 μM palmitoleic acid was diluted into 95% ethanol and dispensed onto the polyurethane films in their respective wells (**Figure 1**).

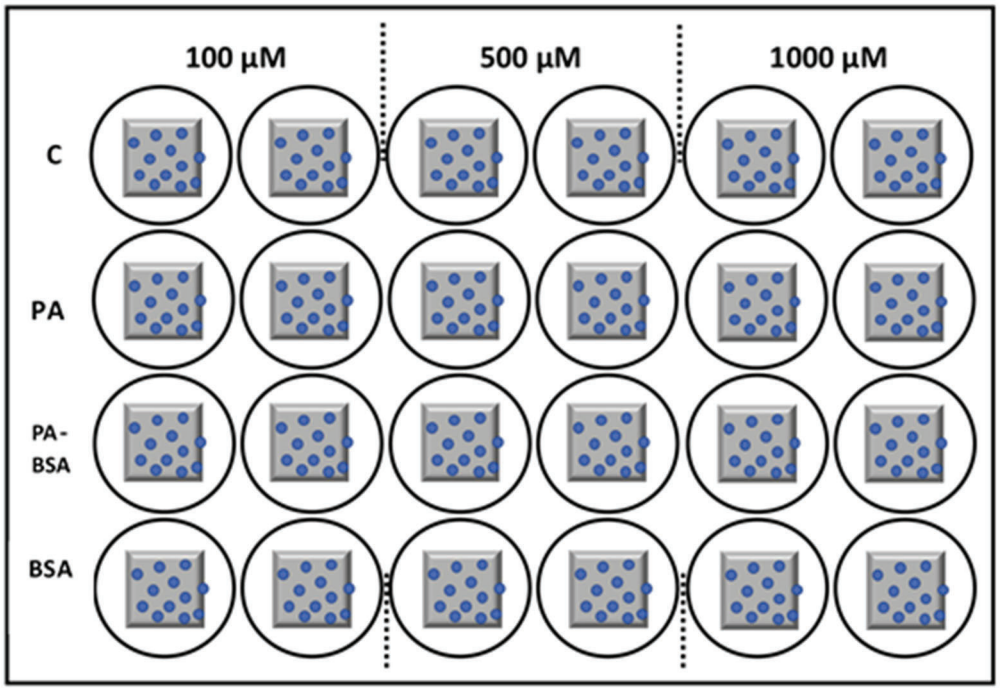

Figure 1. The experimental design consisted of polyurethane films in a 24-well dish. There were two wells per specific treatment and concentration. For each concentration designation (100 µM, 500 µM, and 1000 µM), there were control samples and films coated with palmitoleic acid (PA), palmitoleic acid conjugated with bovine serum albumin (PA-BSA), and bovine serum albumin (BSA), respectively. Ten samples were analyzed for each polyurethane film, resulting in twenty samples per specific concentration and treatment condition.

The control films (C) were coated with NaCl only. All polyurethane films were dried at room temperature overnight to ensure adhesion. For each of the respective BSA wells, the same concentrations of 100 µM, 500 µM, and 1000 µM were added into NaCl instead of ethanol. A ratio of 1 mM of PA to 0.17 mM of BSA was utilized for the wells reserved for the PA-BSA conjugate.[16] For the six PA-BSA conjugate films, the first two films were coated with 100 µM of PA conjugated with BSA. The second pair was coated with 500 µM of PA conjugated with BSA, and the third pair was coated with 1000 µM of PA conjugated with BSA. The conjugate was prepared by stirring the PA and BSA solutions under heat.[16] All polyurethane films were coated with 50 µL of designated solution, then dried at room temperature overnight (in a sterile hood) to ensure adhesion. Any excess coating mixture was removed by pipetting.

These specific PA concentrations were chosen to determine the effects of a low, medium, and high concentration of PA on macrophage attachment to the polyurethane films. Previous research[12] utilized the concentration of 600 µM of PA in cell culture; thus, concentrations around this value were chosen to see the effects of activated THP-1 cell (macrophage) adhesion on the polyurethane films. NaCl was used to ensure macrophage adherence to the control polyurethane films.

C. The THP-1 Attachment Assay

The THP-1 Attachment Assay[13] was utilized in this experiment. All work for the assay was conducted in sterile conditions under a hood. 70% ethanol was used to disinfect any cell culture materials. THP-1 cells purchased from ATCC (Manassas, VA) were utilized. Cells were grown in RPMI-1640 media (Thermo, Denver, Colorado) with 5% FBS (Gibco, Grand Island, New York) and +50 µM 2 - ME (2-Mercaptoethanol; Gibco, Grand Island, New York) at 37 °C and 5 % CO_2 in a Thermo Forma incubator. A minimum of 3 million active growing THP-1 cells were collected and centrifuged at 3,000 rpm. They were counted with the aid of trypan blue and an automated cell counter (Olympus). The cells were deemed acceptable with percent viabilities that were calculated to be above 90%. The cell concentration and the volume of THP-1 cells required to seed 300,000 cells per well were determined. Essentially, the THP-1 cells were activated with Phorbol 12-myristate 13-acetate (PMA) before cell addition into the wells. PMA assists in the differentiation process from monocytes to macrophage by activating protein kinases. Adhesion of differentiated macrophages onto polyurethane films was allowed by a three-day incubation at 37 °C and 5% CO_2. Following this allotted incubation time, the polyurethane films were washed with phosphate buffered saline (PBS) three times, and then the

attached cells were fixed with 1% formaldehyde fixation buffer. After being washed by PBS for three times again, they were stained with Vectashield plus DAPI (Vector Laboratories, Inc) to label the cell nuclei that had attached to the film. The films were analyzed by fluorescence microscopy.

D. Microscopy and Cell Counting

Light micrographs were taken with an inverted phase contrast microscope (Olympus, BX41 and Camera DP73) to view the THP-1 cells after three days of incubation. To further determine the number of adhered macrophages, fluorescence micrographs with the DAPI stain were taken at 100x magnification and analyzed. Ten fields of view were randomly selected and an area between 151,000 and 151,500 μm^2 was utilized for cell counting for each control and experimental film. The selected fields and associated areas were not repeated or duplicated. Twenty sampling areas were analyzed per experimental condition. The adhered number of macrophages were counted by the number of nuclei present and totaled for each field. CellSens software was used to demarcate the specific areas for counting in each field and to assist in the cell counting process. The software allowed markers to be placed on each cell nucleus in each micrograph and total numbers were recorded. Cell count values for each set of two control and experimental films were averaged and compared to determine which group of films experienced the most macrophage adhesion. P values which compared each experimental group to its designated control group were obtained using a GraphPad[17] unpaired t-test with a significance value of < 0.01. Analysis of the p-values influenced conclusions regarding the significance of results.

RESULTS

As displayed in **Figures 2-4,** there was an apparent decrease in macrophage (THP-1 activated cells) attachment to the PA and BSA coated polyurethane films for all concentrations tested (100 μM, 500 μM, and 1000 μM). The PA-BSA coated films presented more adhered macrophages, similar to the control films**.** Ten samples per well (twenty per experimental condition) were analyzed for macrophage adherence per random sampling area (151,000 - 151,500 μm^2).

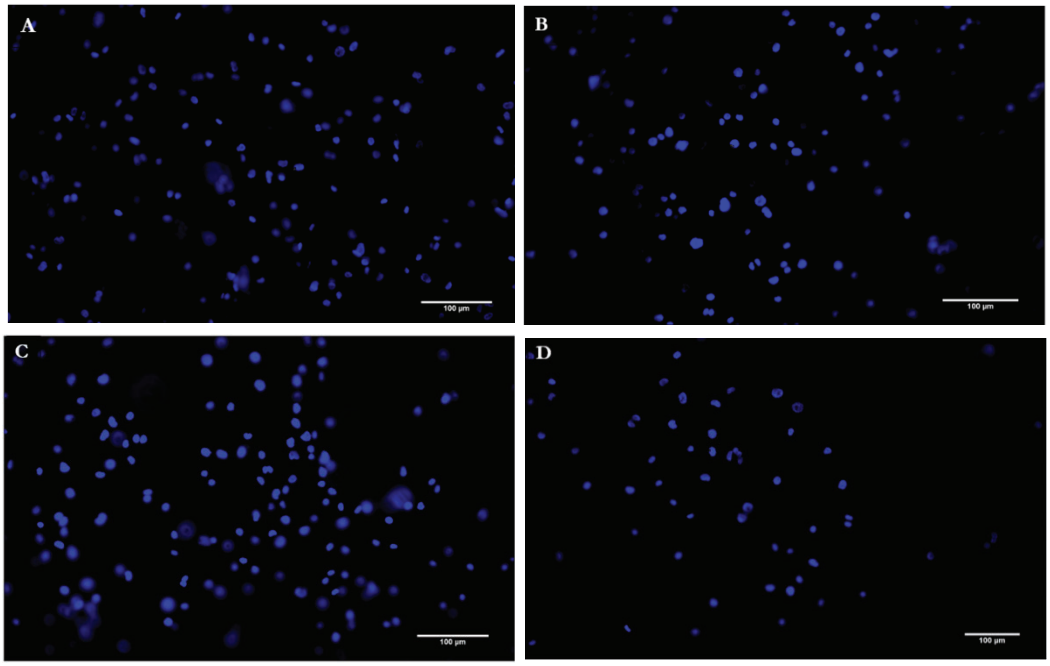

Figure 2. Fluorescence micrographs taken at a magnification of 100X of adhered macrophages (THP-1 activated cells) to polyurethane films coated with 100 μM: NaCl control (A), PA (B), conjugated PA-BSA (C), BSA (D).

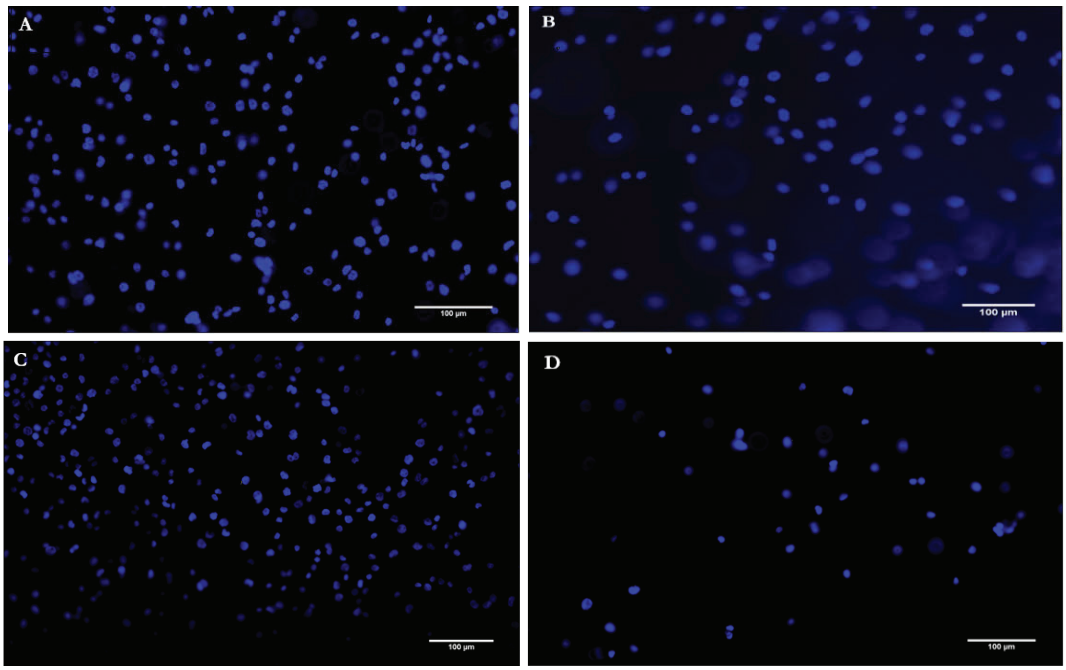

Figure 3. Fluorescence micrographs taken at a magnification of 100X of adhered macrophages (THP-1 activated cells) to films coated with 500 µM: NaCl control (A), PA (B), conjugated PA-BSA (C), BSA (D).

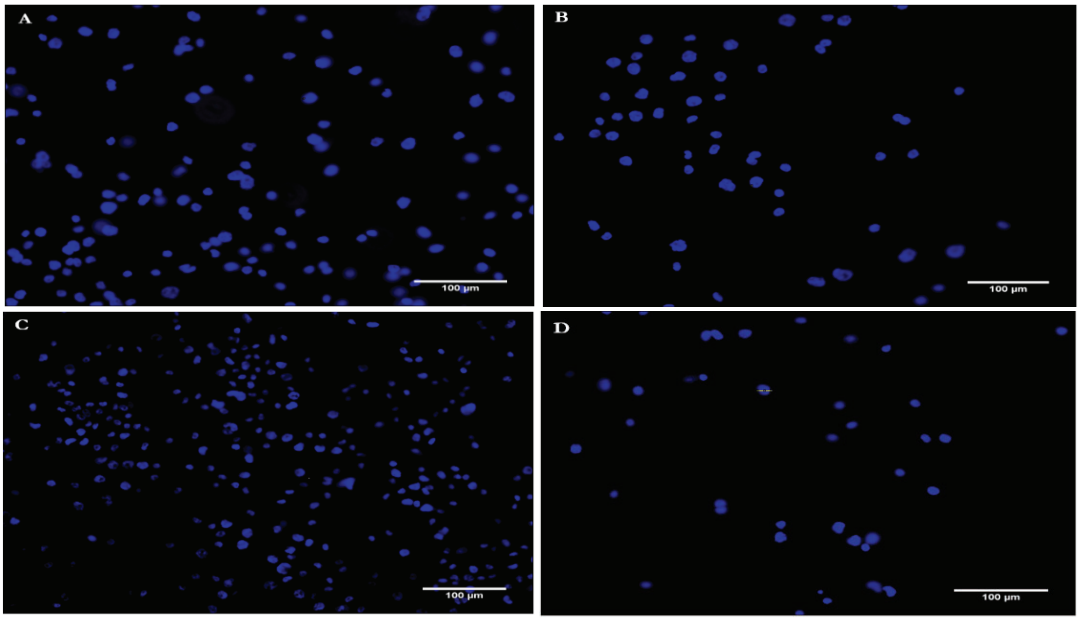

Figure 4. Fluorescence micrographs taken at a magnification of 100X of adhered macrophages (THP-1 activated cells) to polyurethane films coated with 1,000 µM: NaCl control (A), PA (B), conjugated PA-BSA (C), BSA (D).

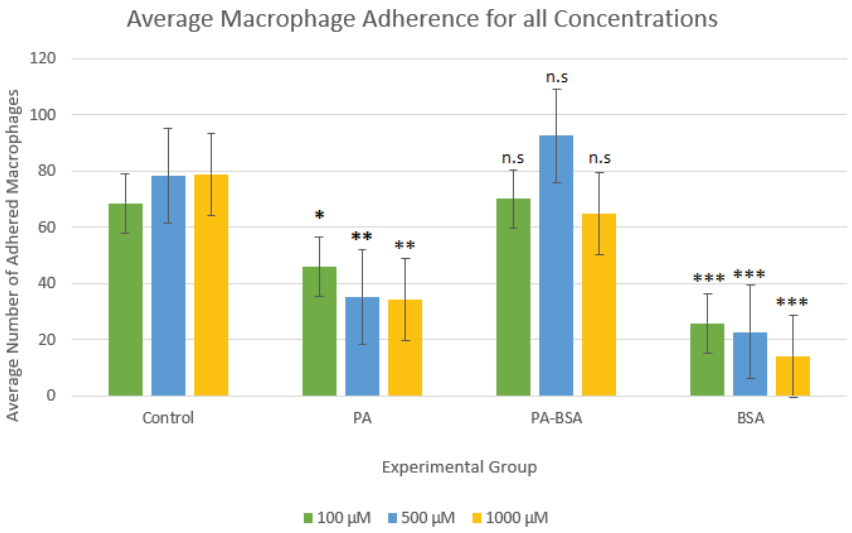

Figure 5. The average number of adhered macrophages per random sampling area (151,000 - 151,500 µm²) in each experimental group showing all concentrations tested: control, PA, PA-BSA, and BSA. Twenty samples were analyzed per experimental condition of concentration and treatment. Error bars are the standard deviations of the mean. P-values were calculated for significance: *p < 0.01, **p<0.001, ***p<0.0001.

The average number of adhered macrophages to polyurethane films with NaCl, 100 µM of PA, PA-BSA conjugate, and BSA are displayed in green bars in **Figure 5**. There was an average of 68.5 macrophages adhered to the polyurethane films with no coating; an average of 45.7 macrophages adhered to the polyurethane coated with PA; an average of 70 macrophages adhered to the polyurethane coated with the PA-BSA conjugate; and, an average of 25.7 macrophages adhered the polyurethane coated with BSA. P-values were calculated for the PA, PA-BSA conjugate, and BSA groups using a GraphPad[17] unpaired t-test with a significance value of less than 0.01. Based on the results, polyurethane films coated with 100 µM of PA and BA exhibited a significant decrease in adhered macrophages.

The average number of adhered macrophages to polyurethane films with NaCl, 500 µM of PA, BSA, and PA-BSA conjugate is presented by the blue bars in the figure. The average number of adhered macrophages per random sampling area (151,000 - 151,500 µm²) for each experimental group was 78.25, 35.05, 92.45, and 22.75, respectively. There was a significant decrease in the number of adhered macrophages on the polyurethane films coated with 500 µM of PA and BSA individually.

The yellow bars in **Figure 5** display the average number of adhered macrophages to polyurethane films with NaCl, 1,000 µM of PA, the PA-BSA conjugate, and BSA. The average number of adhered macrophages per random sampling area (151,000 - 151,500 µm²) on each experimental group was 78.75, 34.15, 64.65, and 14.05, respectively. When 1000 µM of PA and BSA was applied to the polyurethane films, the number of adhered macrophages decreased significantly when compared to the results seen using the 100 µM and 500 µM concentrations.

From these data, it can be concluded that the polyurethane films coated with BSA had the most significant decrease in the number of adhered macrophages compared to that of the control, PA, and PA-BSA experimental groups. PA also caused a significant decrease in macrophage adherence, as apparent by the significant p-values. Both PA and BSA exhibited a reduced number of adhered macrophages to the polyurethane films as compared to the control for all concentrations of 100 µM, 500 µM, and 1000 µM. The average number of macrophages attached to PA coated films were 45.7, 35.05, and 34.15, respectively. The average number of macrophages attached to BSA coated films were 25.7, 22.75, and 14.05. The p-values were less than the significance value of 0.01. On the other hand, the polyurethane films coated with the PA-BSA conjugate presented no significant differences from saline control surfaces as the p-values were much greater than the significance value of 0.01.

DISCUSSION

Studies have shown the damaging effects of the body's host inflammatory response instigated by the presence of implanted medical devices. The presence of implants and the degradation of such implants attract cells of the immune system, prominently differentiated macrophages, that recruit other cells to the affected area by the release of a variety of cytokines and chemokines.[18] This research was undertaken to investigate how the human host response can be reduced by coating such devices with molecules that are not seen as foreign in the body (self-like). The specific hypothesis under investigation was PA, PA conjugated to BSA, and BSA will decrease the number of adhered activated THP-1 cells(macrophages) to polyurethane films *in vitro.* Polyurethane films were chosen since polyurethane is a commonly used polymer in implantable devices and biomaterials. Polyurethanes are produced by reactions between alcohols and isocyanates, the latter of which is considered a 'non-self' molecule in the body. PA, a hydrophobic monounsaturated fatty acid commonly found in adipose tissue, was reasoned because of previous research indicating the anti-inflammatory nature of fatty acids by inhibiting macrophage cytokine production[11]; and, a PA-BSA conjugate was chosen because of BSA's ability to bind hydrophobic fatty acids and efficiency as an aqueous-soluble reagent in cell-based assays.[14] Albumin is a negatively charged, hydrophilic large molecule found in blood plasma, and BSA is a common supplement used in THP-1 suspension cell culture systems. It was assumed that coating polyurethane films with the high concentration of PA, the PA-BSA conjugate, or BSA would exhibit the least macrophage attachment while the control films would exhibit the most macrophage attachment. On the other hand, coating the polyurethane films with the low concentrations of PA, PA conjugated with BSA, or BSA would exhibit intermediate macrophage attachment. The experiment led to contradictory results. Although the hypothesis was supported by the PA and BSA adherence values, the adherence values on films coated with the PA-BSA conjugate across all concentrations were higher than expected.

Data across polyurethane films of all three concentrations supported the hypothesis that coating with PA reduced macrophage adherence *in vitro* as compared to the control films with no coating. However, the BSA films exhibited a significant reduction in macrophage adhesion as well. This indicates that both PA and BSA possess the capability to reduce macrophage attachment. In addition, there was an apparent decrease in macrophage attachment for both substrates as the coating concentration increased. Therefore, the films coated with the higher concentration (1,000 μM) of PA and BSA displayed the lowest adherence values. In addition, data gathered from films of all three concentrations showed that BSA had the least number of adhered macrophages, followed by PA.

Although the results of the PA and BSA individually coated films supported the hypothesis in reducing macrophage attachment to polyurethane films, the results of the PA-BSA conjugate did not. There was no apparent decrease of macrophage adherence on the PA-BSA coated films for all three concentrations. In fact, the data obtained from the cell counts showed that the number of attached macrophages to the PA-BSA films were not statistically different than that of the control films with no coating. In summary, due to these results, the hypothesis under investigation in this study was only partially supported as the PA-BSA conjugate had no repressive effect on macrophage attachment, while the individually coated PA and BSA films did.

This discrepancy regarding the PA-BSA conjugate can be related to the ability of macrophages to recognize "self-like" molecules from "non-self-molecules". It is assumed that by forming the conjugate through constant heating and stirring, a conformational change(s) took place in the PA and BSA conjugate as the fatty acids covalently bonded to the protein.[19] Thus, the activated macrophages proceeded as they are charged to do; they treated the conjugate as a foreign molecule by attaching to it and most likely releasing cytokines and transcription factors that amplified the response. On the other hand, since PA is commonly found in the human body, and BSA in tissue culture for THP-1 cells, the macrophages recognized them as such. This is supported by the increase in number of adhered macrophages to the polyurethane films coated with the conjugate as compared to the reduced number of adhered macrophages on the PA and BSA coated films. With the rise of temperature at pH 7.0, the proportion of alpha-helix decreased above 30 °C and those of beta-structure and disordered structure increased in the same temperature range.[19]

CONCLUSIONS

In conclusion, we found that PA and BSA coated polyurethane films reduce macrophage adherence *in vitro*, while films coated with conjugated PA-BSA had no significant effect. We believe that this research can aid other researchers in finding a viable solution to help slow down the degradation of implanted medical devices composed of polyurethane via activation of the host

response. By exploring the ability of 'self-like' molecules like palmitoleic acid to significantly reduce macrophage adherence, revised manufacturing measures may be investigated to reduce the host response.

For future research, specific cytokine expression released by macrophages in our assays will be evaluated using ELISA and gene expression. We can also test a human peritoneal macrophage cell line instead of THP-1 using a similar experimental design. In vivo experimentation can also be conducted by using animal models. Although this is not the most favorable method, it is vital to the next step.

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ABOUT STUDENT AUTHORS

Sheherbano Hussain is an undergraduate student at Penn State University Park majoring in Biochemistry with a Cell and Molecular Biology option. After graduating in May of 2020 with a B.S. degree, she hopes to apply her skills from undergraduate research for future work in the medical field.

Zoha Babar is an undergraduate student at Penn State University Park. She will graduate in May, 2020 with a B.S. degree in Biology. She plans to pursue a career in the medical field.

Jimmy Hadid is an undergraduate student at Penn State University Park. He will graduate in May, 2020 with a B.S. degree in Biology with a neuroscience option. In the future, he is planning on working in the medical field.

PRESS SUMMARY

The immune system works to defend the body by enacting the host inflammatory response towards foreign materials. Major players in this response are macrophages, which are white blood cells that attack and engulf molecules that are considered 'non-self.' While the host inflammatory response is crucial for protection, it poses a significant issue in healthcare. Since the immune system does not recognize medical implants and prosthetics, macrophages attack the foreign substance, causing deterioration and infection. To suppress the effects of the host response, this research was undertaken to investigate the ability of 'self-like' molecules to reduce activated THP-1 cell (macrophage) attachment to polyurethane, a commonly used polymer in prosthetics. Previous research indicates the anti-inflammatory character of palmitoleic acid in cell culture; and, bovine serum albumin is a common supplement used in tissue culture for THP-1 cells. The reduction of macrophage adherence to polyurethane films coated with palmitoleic acid, palmitoleic acid conjugated to bovine serum albumin, and bovine serum albumin using a tested *in vitro* THP-1 bioassay is presented.