Microglial Dysfunction in Alzheimer’s Disease

Introduction

Alzheimer’s disease is a degenerative neurological disorder which, according to the Alzheimer’s Association, is the sixth leading cause of death in the United States, and will soon be even higher on the list (Alzheimer’s Association). However, there currently is no effective (or even realistic) treatment for Alzheimer’s, in part due to science’s vast lack of knowledge about the biology, pathology, and etiology of the disease. Psychologically, Alzheimer’s disease is characterized by increasingly severe cognitive deficits and functional impairments (American Psychiatric Association, 1994). Biologically, the disorder is manifested by the presence of amyloid-beta plaques and neurofibrillary tangles throughout the brain, as well as progressive, brain-wide, neuronal death, typically beginning in the temporal lobes. Many researchers presume that these biological hallmarks of Alzheimer’s disease trigger this neuronal death, but causality has yet to be determined (Chamberlin & Narins, 2005).

Some neurodegeneration researchers modify or reject the plaque and protein hypothesis in favor of other theories. The second major hypothesis that exists, the “inflammation hypothesis,” posits that inflammation in the central nervous system (CNS) is a major determinant and exacerbating factor of neurodegeneration, which leads to Alzheimer’s disease (Zotova, Nicoll, Kalaria, Holmes, & Boche, 2010). This hypothesis is intricately related to the “microglial dysfunction hypothesis,” which suggests that it is not the increase of inflammation which is implicit in Alzheimer’s disease, but rather the dysfunction (or loss of function) of neuroprotective microglial cells in the CNS that triggers it (Polazzi & Monti, 2010). Both of these hypotheses rest upon the idea that the branch of the immune system in the CNS, the microglia glia cells, is dysfunctional in Alzheimer’s disease.

The immune system is composed of a variety of cells tasked in protecting the human body from invasion and infection from outside agents. This system is divided into the peripheral immune system and neurological immune system, as the blood brain barrier (BBB) was thought to be impermeable to peripheral immune cells. However, this distinction is rapidly being blurred, as current research has demonstrated that the permeability of the BBB can be altered and increased in certain disease states (Takeda et al., 2013). In the periphery, this system is divided into three main division—nonspecific immunity, humoral immunity, and cell-mediated immunity. In nonspecific immunity, leukocytes enter an area of infection and release chemicals, such as histamines, which recruit more leukocytes and increase blood flow to the affected areas, triggering inflammation. This inflammatory response occurs due to injury or invasion in an early attempt to stave off further infection. Humoral immunity is mediated by B-cells. These cells produce antigen-specific antibodies, which bind to their target antigen, neutralize it, and tag it for destruction by other circulating leukocytes. Cell-mediated immunity is a function of T-cells, which attack and destroy tagged foreign pathogens through mechanisms including phagocytosis and the release of cytotoxic molecules (including interleukins and cytokines) (Campbell & Reece Biology, Chapter 43).

Within the CNS, immune system function works though derivatives of innate and cell-mediated mechanisms by the action of microglia. Microglia are glial cells which make up about 10% of the mass of the CNS
(Solito & Sastre, 2012). These cells are unique in that they have the capacity to change phenotype due to the local microenvironment. In healthy adults, the majority of microglia are in the ramified, or undifferentiated, state. This phenotype is ready to morph when an altered environment is detected. Ramified microglia pseudopodia sense the microenvironment for threats in the CNS. If one is detected, the ramified microglial cell will become activated and will either take the M1-like or the M2-like phenotype (the names are derived from the similarity of the activated states of peripheral macrophages). What phenotype the microglia takes depends upon the composition of the microenvironment (Olah, Biber, Vinet, & Boddeke, 2011). M1 phenotype microglial cells are largely neurotoxic. When microglia are in M1, they release toxins, such as reactive oxygen species (ROS), cytokines, and other cytotoxic chemicals, that can cause damage to neurons. Microglia in this phenotype work in very much the same fashion as cytotoxic T cells, as they attack and neutralize foreign antigens to prevent infection. When the microglia detect LPS, Tumor Necrotic Factor alpha (TNF-α), or interferon-γ, they morph into the M1 phenotype, and trigger the inflammatory response. M2 phenotype microglial cells are largely neuroprotective. When microglia are activated in this state, they release anti-inflammatory compounds and neurotropic factors (molecules which support the growth of neurons). When ramified microglia detect Interleukin-4, Interleukin-13, glucocorticoids, Tumor Growth Factor Beta (TGF-β), or Interleukin-10, they morph into M2. These chemicals help to foster neuron growth and metabolism (González, Elgueta, Montoya, & Pacheco, 2014).

When an insult to the CNS is detected, undifferentiated, ramified microglia and associated macrophages migrate toward the location of the injury and begin releasing histamines and related compounds, triggering the inflammatory response (Członkowska & Kurkowska-Jastrzębska, 2011). Upon reaching the area of insult, the ramified microglia will taste the microenvironment and will be activated to either M1 or M2 phenotype. From there, the cell will remain activated until the disturbance is ameliorated, and will either remain in the current phenotype, revert to the ramified phenotype, or switch phenotypes. Chronic neuroinflammation and, potentially, neurodegeneration, thus can occur when microglia remain activated in the M1 phenotype.

The Microglial Dysfunction Hypothesis presumes that the lack of undifferentiated cells is a major contributor to neurodegeneration. There is ample evidence indicating that in many neurodegenerative diseases, microglia remain hyperactivated, leading to chronic inflammation in the brain (for review, see Khandelwal, Herman, and Moussa (2011)) or to biological hallmarks, such as tau proteins. Kitazawa, Oddo, Yamasaki, Green, and LaFerla (2005) studied the effect of chronic neuroinflammation in Alzheimer’s pathology. They triggered neuroinflammation in six 3xTg-AD transgenic Alzheimer's disease model mice by injecting lipopolysaccharide (LPS) into their intraperitoneal cavity repeatedly over a period of six months. LPS is an endotoxin which travels across the BBB and stimulates microglial activity. They also injected the same concentration of LPS into non-transgenic mice for the same period of time as a positive control. The researchers measured the effect of neuroinflammation by doing immunohistochemical staining for Aβ and tau protein deposits for each mouse. Moreover, Kitazawa et al. completed a cdk5 (cyclin-dependent kinase) assay for each of the twelve mice to determine if its activation had any bearing on pathology, given the fact that riscovitine (an inhibitor of this kinase) staved off neuroinflammation. The logic behind this experiment is that if the presence of either of the proteins is increased across all mice in the transgenic condition and not in the control, then inflammation itself exacerbated the
disease in the already genetically predisposed mice. Moreover, if cdk5 levels were increased in the mice that had higher levels of either protein, then cdk5 activation would be a mediator in this response. The researchers found that both cdk5 and hyperphosphorylated tau protein aggregate levels were greatly raised in the LPS condition. Thus, inflammation (caused by microglia and mediated by cdk5 activation) exacerbates Alzheimer’s pathology.

A second line of evidence implicating the importance of neuroinflammation in Alzheimer’s comes from research examining the effect that nonsteroidal anti-inflammatory drugs (NSAIDs) have on the development and progression of the disorder. Research has shown that long-term NSAID use delays the onset of Alzheimer’s, both in mouse models and, anecdotally, in humans (Lim et al., 2000). Lim et al. (2000) further studied this idea by administering NSAIDs to transgenic Alzheimer’s mice over a period of time and examining microglial activity and levels of Aβ deposits. The researchers took two strains of mice (transgenic [TG+] and non-transgenic [TG-]) and fed them chow with or without NSAIDs for a period of six months. After slaughtering the mice, they performed a Sandwich ELISA and immunostained for Aβ to determine the levels of this protein in each mouse’s brain. They also did a quantitative morphometric image analysis of anti-PT labeled microglia to determine the level of activation of microglia cells within each condition at the time of death. The logic behind this experiment is that if levels of Aβ are lowest in the TG+NSAID condition, then NSAID use can reduce Aβ pathology. Moreover, if levels of staining of anti-PT labeled microglia are concurrently lower in the TG+/NSAID condition, then the reduction in Alzheimer’s pathology is a result of the anti-inflammatory properties of the NSAID. In fact, this is exactly what the Lim et al. found. TG+ mice that were given steady diets with ibuprofen additives had significantly reduced microglial activity and depleted Aβ deposits. They hypothesized that the NSAIDs reduce the neurotoxic effects of the microglia (hence reducing M1 phenotype microglia activity). While NSAID use in humans has failed to produce results (and was extremely toxic), the idea that reducing neuroinflammation as a preventative measure of Alzheimer’s disease (but not a method of disease reversal—see Yu et al. (2012)) is certainly a plausible and worthy one (Khandelwal et al., 2011).

The present study strives to fully test the idea that the reduction of inflammation can be a protective measure against Alzheimer’s disease. As previously described, using NSAIDs as a method of anti-inflammation is ineffective in humans. Not only is it an omnibus inflammation reducer (which is physiologically necessary and beneficial if controlled), but the drug is toxic to the body in with extended use. Thus, a new mechanism that targets specifically M1 microglia that cause chronic inflammation, using a drug that is not toxic, is necessary. Therefore, in this study, we will target differential activation of microglia. As previously described, microglia have two phenotypes—one neurotoxic (M1) and one neuroprotective (M2). The logic behind the present experiment is that if we can switch microglia phenotype from M1 (which is typically found in higher concentrations in Alzheimer’s disease, specifically surrounding Aβ plaques) to M2 (which promotes neuron growth and metabolism), then Alzheimer’s pathology will be reduced or delayed.

We aim to trigger M1 morphology in both ramified- and M2-phenotype microglia through competitive inhibition. In transgenic (Tg+) Alzheimer’s mice (and control non-transgenic [Tg-] mice), we will insert the drug hydrocortisone (or a control Artificial Cerebrospinal Fluid [ACSF]) into the cranial cavity during the first six months of the life of the mice. As hydrocortisone (a glucocorticoid) has been shown to trigger the M2 phenotype, we
hope that, with repeated doses of the drug, endogenous M1-triggering molecules will be outcompeted (González et al., 2014). This will facilitate more microglia to enter the M2 phenotype. We will also use the Morris Water Maze weekly to test the cognitive effects of the treatment (see Puzzo, Privitera, and Palmeri (2012)). After six months, we will sacrifice each mouse and use immunohistochemical techniques on the cultured brain tissue to measure the ratio of M1:M2:Ramified microglia. There will be four conditions in the experiment: Transgenic/glucocorticoid (Tg+/glucocorticoid), Transgenic/Artificial Cerebrospinal Fluid (Tg+/ACSF), Non-transgenic/glucocorticoid (Tg-/glucocorticoid), and Non-transgenic, Artificial Cerebrospinal Fluid (Tg-/ACSF). The critical condition in this experiment is the Tg+/Glucocorticoid condition. If, in this condition, the levels of activation of M2-phenotype microglia are significantly higher and if the latency to find the platform in the Maze is significantly less than the control Alzheimer’s mouse, Tg+/ACSF, (and not significantly different to the normal mouse, Tg-/ACSF), then this treatment, and thus treatment philosophy, is effective. Because the Tg-/ACSF condition is functionally equivalent to the critical condition, latency results and the M2 phenotype activation levels of this condition should not be significantly different from the critical condition. In the Tg+/ACSF condition, we expect to see high latency in the maze as well as high levels of M1 phenotype microglia, as these mice should exhibit Alzheimer’s symptomology. In the Tg-/glucocorticoid condition, we expect to see an overall lower level of M1 phenotype and higher level of M2 phenotype microglia than the baseline control (Tg-/ACSF), as hydrocortisone (the glucocorticoid) should stimulate M2 phenotype microglia. If these results are found, then this treatment mechanism (i.e. competitive inhibition to trigger M2 phenotype of microglia), can potentially be an effective treatment option for Alzheimer’s disease.

Methods

Subjects. 12 3xTg-AD transgenic mice (Tg+) were used. These mice have three mutant genes (APPK670/671N, PS1
M146V, and tauP301L) and develop the hallmarks of Alzheimer’s disease (Aβ and tau pathology as well as cognitive
effects) as they age, typical of a human Alzheimer’s patient (Kitazawa et al., 2005). Additionally, 12 normal, non-
transgenic mice (Tg-) were used.

Glucocorticoid Manipulation. The glucocorticoid hydrocortisone was inserted into the cranium of six Tg+ mice and
six Tg- mice via an IV catheter. To the other six Tg+ mice and six Tg- mice, equivalent amounts of Artificial
Cerebral Spinal Fluid (ACSF) were inserted into the cranium to control for surgery and injection effects. Injections
of 0.2mg/kg of body weight of hydrocortisone were made once a day for four months. The dosage was calculated by
the minimal dose of hydrocortisone recommended for adults (Drugs.com). The equivalent concentration of ACSF
was injected to the remaining mice. Thus, a 4 x 4 design was utilized, in which both Tg+ and Tg- mice received both
glucocorticoid and ACSF.

Behavioral Testing. The Morris Water Maze was used to test cognitive decline in the mice. At the outset of the
experiment (before any injections but after the surgery), mice were trained twice a day for a week in the Maze to
ensure that they used long term memory to find the platform in the trials during the experiment. The time to find
the hidden platform (latency) was measured for each trial. Training ended when all mice had an equal starting latency
(Puzzo et al., 2012). After training, during the injection/test period, the water maze was filled with an occluded
liquid so as to hide the platform to ensure that the mouse was relying solely on memory and not their sensory
systems. The mouse was placed in the water maze at the same location in each trial, and the amount of time it took for the mouse to find the platform (latency) was measured. Each mouse ran the maze one time every seven days (i.e. each mouse had a scheduled weekly maze time). Maze time was scheduled 3 hours after injection. The mice ran the maze weekly from the beginning of injection at 2 months of age until they were sacrificed at 6 months.

Immunohistochemical Staining. At six months of age, each mouse was sacrificed and their brains were harvested for testing. We were interested in determining the relative ratio of M1:M2:Ramified phenotype microglia in each of the conditions. To do this, we fluorescently tagged a different membrane protein on each of the microglia phenotypes. We first sliced each of the brains and placed them in ACSF with HEPES buffer. After suctioning the ACSF/HEPES solution out of the preparation, we washed each slice with primary antibodies to a protein specific to the M1 phenotype microglial membrane (this protein has yet to be discovered). After letting it sit in the antibodies for 30 minutes, we suctioned out the solution and washed the slices with ACSF/HEPES. Then, after removing the rinse, rabbit secondary antibodies to the primary antibodies (which are fluorescently tagged red) were added and set to incubate for 45 minutes. After those 45 minutes, the secondary antibodies were removed and the slices were again rinsed with ACSF/HEPES. This procedure fluorescently tagged M1 phenotype microglia red. To tag M2 phenotype microglia, we utilized the same procedure on the same brain slices, using a different primary antibody. We first (after removing the storage ACSF/HEPES solution) incubated the slices with primary antibodies to a membrane protein specific to the M2 phenotype microglia (which also has yet to be discovered). Then, after 30 minutes, we washed the slices with ACSF/HEPES and added the rabbit secondary antibodies to the primary antibodies (which are fluorescently tagged yellow) and let them incubate for 45 minutes. After that time, we suctioned the secondary antibodies and put the slices back into a solution of ACSF/HEPES. This procedure allowed us to tag M2 phenotype microglia yellow. Assuming ramified microglia have both of these proteins on their membrane (again, has not yet been discovered), then the ramified phenotype will be detected by an orange fluorescence (the combination of yellow and red).

Analysis. We examined the levels of fluorescence of each of the three colors (yellow, red, and orange) through using a fluorimeter. Within conditions, we took a mean level of fluorescence across all mice to get a "typical" level of activation for each condition. Then, still within conditions, we ran an ANOVA to determine whether any of the phenotypes were more activated. Between conditions, we ran an ANOVA for each level of fluorescence to determine whether the levels of activation of each of the phenotypes were different for each condition. We also examined the latency of finding the platform in the Morris Water Maze. We first examined the mean latency across conditions and ran an ANOVA to determine if there were any significant differences. Hypothetically, the mean of the Tg\(^+\)/glucocorticoid condition should be lower (i.e. they found the platform faster) than the mean of the Tg\(^+\)/ACSF condition, as in this condition, the mice lose their memories as the disease progresses. We also ran a regression analysis of time of trial vs. latency for each condition. Theoretically, only the Tg\(^+\)/ACSF condition should show an increase in latency. The rest should decrease or remain the same as a result of learning effects. Finally, we correlated the mean latency of each condition and the level of activation of M2 phenotype microglia to determine if there is any relationship between M2 phenotype microglia activation and cognitive preservation.
ENGENDERING MEXICAN MIGRATION STUDIES: THE POTRAYAL OF MIGRANT MEXICAN WOMEN IN SCHOLARSHIP AND THEIR AGENCY WITHIN TRANSNATIONAL MIGRATION DURING THE TWENTIETH CENTURY

History 487: Immigration in the U.S.
Term Paper Excerpt
When treating gender as a central organizing principle in immigration studies, a multitude of topics and inquiries emerge, which include but are not limited to: how and why immigrant women and men have different experiences when migrating and what role women have played in transnational migration (a topic that, for some time, was not gendered in scholarship and was notorious for equating “migrant” with “male”). When investigating the role that Mexican women played in transnational migration during the twentieth century, in particular, the historical inquiries noted earlier become incredibly significant despite not having received scholarly attention until the latter decades of the twentieth century. With the rise of women’s migration from Mexico to the United States during the nineteenth and twentieth centuries, Mexican women inevitably became entangled in a complex and dynamic borderland where internal and international migration processes converge: the U.S.-Mexico border. During the twentieth century, most notably, Mexican women who engaged in internal and international migration facilitated by the involvement of women in formal-sector employment, changing family dynamics and gender roles, immigration policies attempting to improve family unity, and patterns of recurrent male migration demonstrated that women (1) subtly and visibly contested oppressive gender roles while making modest gains by using migration as a survival technique and means with which to achieve emancipation and (2) exhibited a demonstrable amount of agency in the transnational migration of Mexican individuals to the U.S..

According to Patricia R. Pessar’s historiography of gendered migration studies, she notes that women, central agents worthy of thorough investigation, were largely absent from scholarship of immigration history until the mid-1970s. She claims that most historians were

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2 Ibid.
influenced by traditional theories in which migrants were overwhelmingly characterized as “Western [men]” pursuing modern, city lives. Because this journey required that its undertakers be risk-takers and high-achievers, ostensibly only men fit the criteria. On the other hand, women, if portrayed in these narratives and studies, were depicted as “guardians of community tradition and stability.” Women were strictly mothers who did not venture out into the unknown. Instead, they stayed at home and maintained the stability of their restrictive space: the domestic sphere. If immigration scholars did not subscribe to this “neoclassical theory,” they subscribed to notions that only male immigrants’ lives were worthy of analysis and documentation or that the history of male migrants was gender neutral, making it unnecessary to introduce female players to the stage. The lack of attention paid to women in migration scholarship and their later emergence onto the academic scene of immigration history and studies is significant because it seems to reflect the same resiliency and contestation of roles that twentieth-century Mexican women engaged in to make themselves visible by heading their own households, enabling the migration of other men, and working in public-sector employment. Understanding Mexican immigration to the U.S., then, becomes more valuable when done with a gendered approach.

In using a gender optic to analyze the transnational migration processes that converged on the U.S.-Mexico border, it is important to understand the motivations that prompted Mexican women’s increasing immigration. First of all, it is crucial to point out that Mexican women have had a long history of working outside the home, contrary to both popular belief and recent ethnographic and historic scholarship. In part, the reason why Mexican women’s work outside

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of the home was not being documented was because it was often invisible to the formal census. Mexican women’s work, particularly in the nineteenth and early twentieth centuries, consisted of running a small store from their own homes, sewing garments, selling various items door to door, or raising livestock. To demonstrate quantitative evidence of women’s early employment scarcely reported by other early twentieth century scholars, anthropologist Tamar Wilson notes, “In 1811, women eighteen years of age or older constituted 30.9 percent of the capital city’s labor force; by 1848 this percentage had risen to 36.6 percent and may be underestimated since indigenous women coming into the city to vend their wares may not have been counted... A survey of the period from 1876 to 1970 (Thompson 1991) reveals... There was a peak from 1850 to 1882, a trough from 1920 to 1940, and then a continual increase in women’s labor-force participation thereafter, becoming marked in the 1970’s.”

With the help of these statistics, Wilson is trying to show that women’s participation in Mexico’s labor force, although generally undocumented in early migration scholarship, was, in fact, existent and, more importantly, significant. Women’s increased participation in the labor force was ultimately facilitated by a number of circumstances: rising levels of educational attainment, declining fertility, internal migration from rural to urban areas within Mexico, and a rising proportion of female-headed households. Women ultimately desired to make substantive contributions to maintain their own and their family’s well-being whether they had become the head of their household or not. This was especially true during times of difficult and detrimental economic crises where additional earnings were necessary to supplement the household income.

The rise of women’s employment helped give way to the transformation of historic gender relations governed by law. Early in the history of the treatment of Mexican women, women’s rights were subject to patria potestad in which fathers had full control over their wives.

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6 Tamar Diana Wilson, Women’s Migration Networks in Mexico and Beyond, Albuquerque: University of New Mexico Press, 2009, 2.
7 Ibid.
8 Ibid, 9.
and single children. Under this legal practice, women constantly faced disininheritance, imprisonment, and even death if they were to commit adultery or otherwise disgrace the honor of their fathers or husbands. Eventually, the Civil Codes of 1870 and 1884 repealed the practice of patria potestad and afforded women new sources of autonomy with which to leave their home, have authority over their children, to separate from husbands for justifiable reasons, and acquire property. Other freedoms women eventually gained in the twentieth century through institutional and legal acceptance were divorce and the right to vote.

Even with these changes in the law, which legally shifted gender relations to afford women heightened autonomy as individual agents possessive of their own rights, the effects of machismo, domestic violence, and blatant infidelity would still persist. This brings light to some historian’s views (like that of Patricia Pessar’s) that while Mexican women, over time, have gained a number of significant freedoms and strides towards gender equality, their gains should be viewed as modest. The reasoning for this, in part, involves the economic contributions women attempted to make to their households with their emerging presence in the public sphere as employees during the twentieth century. Despite women’s desire to contribute positively to their household and their obvious efforts to realize this goal, “Women who worked in factories and workshops alongside men were [still] considered outside the normative moral order and essentially morally stigmatized (Porter 2003, chap. 2). Employment of wives, mothers, and children in the multinational assembly plants does not necessarily lead to women’s empowerment; this is partially because it is their family’s economic vulnerability that drives

9 Wilson, Women’s Migration Networks in Mexico and Beyond, 9.
10 Ibid, 9-10.
them into the work force (Fernández-Kelly 1983, 137, 192).”¹² So while, legally, women were making large strides in gender equity, on the ground level, they still experienced moral stigmatization and the impacts of machismo, domestic violence, and infidelity.

It is important to make clear that in highlighting the evidence of modest gains women made in the twentieth century (contrary to the absolute and enthusiastic claims of some historians and other scholars who asserted that women achieved unequivocal empowerment as a result of their improved educational and employment opportunities and subsequent presence in these areas), this is not an attempt to minimize or demean the gains women have achieved in the arena of gender relations. Rather, it is an attempt to raise awareness of why women’s vocal/subtle contestation of the oppressive gender roles they still faced well into the mid-twentieth century necessitated the use of migration as a visibility-augmenting, survival technique.

¹² Wilson, Women’s Migration Networks in Mexico and Beyond, 7.
Fabry-Perot Observations of Lunar Exospheric Potassium Emission

Abstract - The lunar atmosphere, a thin, loosely bound exosphere, must be continually replenished in order to offset its constant loss of constituent vapors to space and surface re-capture. Influenced by interactions between the lunar surface, solar wind, solar radiation, and meteoroid impacts, it exists in a variable equilibrium. Study of its sources and sinks can yield valuable insights into space weather. Spectral line profiles of sodium and potassium can be derived from ground-based observations in the elements’ respective wavelengths. The lines’ widths and areas can be converted to direct measurements of the temperature and column density of the lunar exosphere. High signal-to-noise widths and areas of the potassium D1 (7699 Å) line, produced from lunar exospheric potassium observations, are presented here. Taken at various latitudes and altitudes around the moon using a ground-based Fabry-Perot interferometer between January 2014 and 2015, the data show potassium temperatures and densities before full moon to be consistently hotter and thinner than afterward, though more analysis is needed to confirm the trend.

INTRODUCTION

Potassium and sodium have offered a valuable window into the workings of the lunar atmosphere since the discovery of their presence in 1988 (Potter & Morgan; Tyler et al.). Unlike the earth’s bounded atmosphere, the lunar atmosphere is a tenuous exosphere: it must be continually replenished in order to offset the constant depletion of its constituent vapors to gravitational loss and recycling to the surface (Stern 1999). While these two elements represent only a trace portion of the lunar regolith, they are emitted from the lunar surface at relatively high rates, making them major components of the lunar exosphere (Sarantos et al. 2010). As such, they are important indicators of the interactions between the lunar surface, the solar wind (a stream of charged particles flowing outward from the Sun), solar radiation (radiant energy emitted by the Sun), and meteoroid impacts. They provide a convenient method of studying space weather without the interference of the terrestrial magnetic field, which inhibits efforts to do so using near-Earth satellites. While this opportunity has spurred more than a quarter century’s worth of observation and modeling, the vast majority of data collected and analysis performed has been in the realm of sodium.

There are a variety of source mechanisms by which sodium is released from the lunar surface, but the main contributors are micrometeoroid impact vaporization and photo-stimulated desorption (PSD). During the former, small meteoroids strike the moon and eject sodium from its surface into the exosphere; during the latter, solar ultraviolet radiation destroys the bonds tying sodium atoms to their positions in the lunar regolith. Impact vaporization, an extremely energetic process, produces gases of much higher temperatures (3000 K) than PSD (1200 K) (Sarantos et al. 2010). Measuring the temperature of the sodium exosphere can therefore reveal the relative rates of the two processes at a given time and position on the moon. Atoms in a hot lunar gas are likely to have been liberated from the surface through impact vaporization, while those of a cool gas were probably freed predominantly through PSD. Furthermore, it is currently conjectured that, although PSD rates vary as a function of lunar phase angle and time, impact vaporization rates remain essentially constant throughout the lunar cycle (Sarantos et al. 2010). This indicates hot exospheric gases should be thinner than their cold counterparts, as high relative rates of impact vaporization demand small absolute rates of PSD and, therefore, small overall rates of liberation.

Recent models indicate that, through ion sputtering, the solar wind primes sodium in the lunar surface to be more easily liberated from the regolith by PSD (Sarantos et al. 2010); the sodium atoms can then be driven away from the lunar surface by solar radiation pressure. This priming mechanism is interrupted during full moon, when the moon passes through the earth’s magnetotail (Sarantos et al. 2010). At this time, the lunar surface is shielded from the solar wind, but bombarded by plasma sheet ions. In the case of sodium, this bombardment has two effects. The plasma sheet ions appear to less efficiently prime the sodium in place of the solar wind; sodium emission during the full moon is correspondingly fainter and, thanks to the increased share of impact vaporization liberation, hotter (Potter et al. 2000). In addition, this ion priming appears to increase the solar wind’s effects once the moon exits the magnetotail, leading to increased and cooler sodium emission after full moon.

Because the collection of potassium data is much smaller than the set of sodium data, potassium behavior has not been so extensively modeled, though enough evidence exists to draw some conclusions. The plasma sheet ion priming of potassium, unlike that of sodium, is not strong enough for potassium to be liberated from the lunar regolith while the moon is passing through the magnetotail (Potter et
al. 2000). As a result, potassium emission ceases during the full moon. While there are also some indications that potassium emission increases after the moon exits the magnetotail in the manner of sodium (Sarantos et al. 2010), data to this effect has been extremely limited. This proposed increase is supported by the data presented here, which compares the intensity and temperature of potassium emission in observations from before and after the cessation of potassium emission during full moon.

**Literature Review**

The discovery of lunar sodium and potassium represented a revival of interest in the lunar exosphere. Some of its chemical makeup had been known since the days of the Apollo program (Hodges et al. 1974), although models of the time made the incorrect assumption that the gases existed in thermal equilibrium with the surface. Following the discovery of sodium and potassium emissions from Mercury, researchers spent several years unsuccessfully seeking the same results from the moon (Hunten et al. 1988). The emissions’ eventual discovery in 1988 by Potter & Morgan (followed shortly by Tyler et al.) came by observing along a path just beyond the moon’s sunlit limb, thereby boosting the emissions’ feeble intensities (Sprague 1992).

This method has continued to yield results in the intervening years, prompting a corresponding interest in numerical modeling and theory, though mostly in sodium work. As previously stated, models have posited that lunar sodium is liberated through PSD after being primed by the solar wind (Sarantos et al. 2010). The solar wind’s effects are increased following the lunar passage through the magnetotail due to plasma sheet particle bombardment’s enhancement of the subsequent priming effects of the solar wind (Potter et al. 2000). While Sarantos (2010) surmises that potassium priming mechanisms may act in much the same manner, there have been no models offered to fully explore this possibility, and although he has attempted to define a latitude-dependent underlying behavior (ignoring variations with regard to the lunar cycle), collected data has not yet yielded a consistent morphology against which theory may be tested (Stern 1999).

**Procedures**

Several observing runs were conducted at the National Solar Observatory McMath-Pierce Solar Telescope (MMP) on Kitt Peak, Arizona. Each run consisted of several nights, usually arranged so that full moon fell roughly halfway through the run. This approach yielded observations at lunar phase angles stretching from 40° (the point at which potassium emission appears to become undetectably low due to magnetospheric interference) to 90°. One observing run in January 2015 was conducted over five nights surrounding Third Quarter, yielding observations at phase angles from 120°-160°. A corresponding run centered around First Quarter yielded no data due to unfortunate weather.

Observations of the potassium D1 line (7699 Å) were made with a water-cooled Andor iKon CCD camera and a high-resolution, dual 50 mm-etalon Fabry-Perot (FP) interferometer with a resolving power of ~180,000 and a 3 arcmin (0.2 lunar radii) field of few. The observations were located at specified lunar latitudes by moving the MMP field of view along a cardinal direction from various craters to just beyond the moon’s sunlit edge, corresponding to an altitude of 180 km above the lunar surface. Figure 1 details a typical potassium FP exposure. The data show intensity as a radial function of wavelength, so that rings near the center represent light with shorter wavelengths than rings at larger radii. The bright ring in the center of Figure 1 is lunar emission at the potassium D1 line, positioned inside a wider, dark ring: solar Fraunhofer absorption at the same wavelength.

Due to the unique structure of the MMP, significant amounts of scattered light were introduced to the observations. This created an irregular continuum in each image that increased error and made clean spectral line profile production difficult; the background can be seen in Figure 1, growing sharply darker from left to right. To combat this effect, the continuum was iteratively fit and removed from the image using modeled potassium data and a fifth degree, two-dimensional polynomial to represent the scattered light. Figure 2 demonstrates the procedure for the image from Figure 1. The result contains emission and absorption rings on a flat background of low-amplitude fringes, which represent a characteristic pattern of the CCD chip and have only a miniscule effect on the resultant data.

**Figure 2: Background Subtraction Algorithm**

The background-subtracted images were subsequently converted to spectral line profiles by binning data in concentric rings of equal area, a well-proven technique in FP analysis (Coakley et al 1996). The first data point in the spectrum corresponds to the mean value of a circle of pixels at the image’s center, while each successive data point represents the average value of the pixels in a concentric annulus. A representative spectral line profile from January 2015 is shown in Figure 3. Each spectrum is fit with a three-component Voigt profile containing a negative-area feature (representing the Fraunhofer absorption) and two linked
positive-area features (representing the potassium D2 doublet). The data is fit to the profile using Carey Woodward’s VoigtFit software suite, which accounts for the FP instrumental profile (IP) by convolving the model and IP at each step of the fit process. This yields deconvolved centroid, width, and area values for each observation, which in turn represent direct measurements of the radial velocity, temperature, and column density of the lunar potassium exosphere at the observation’s altitude and latitude. This Voigt fit line is included in Figure 3.

Preliminary Results

Two plots (“Area” and “Width”, with respective figure letters “a” and “b”) from each observing run are presented in the Appendix. Data points, representing observations, are plotted with a symbol denoting the crater with which the MMP field of view was set. Symbols are filled only if the observation was made after full moon, during Third Quarter.

The area of each observation’s spectral line profile is plotted against its lunar phase angle. If two points at the same phase have drastically different areas, the observation with higher area was likely made during a period of increased potassium liberation, although care must be taken to account for atmospheric extinction and a proposed latitudinal dependence. In this work, these effects are ignored, and areas are presented only for rough comparison. Each observation’s spectral line width is also plotted against its lunar phase angle. The conversion from width to temperature is robust and known to be independent of lunar latitude or atmospheric extinction, so equivalent temperature lines have been placed on the plots to better communicate the observations’ measured temperatures.

The width data appear to confirm the proposed priming of potassium liberation during the moon’s transit of the terrestrial magnetotail. Months with solely post-full moon observations (April 2014 and January 2015) show consistently lower temperatures than months with pre-full moon observations such as December 2014. Furthermore, months with both pre- and post-full moon observations, especially February and May 2014, show a clear temperature separation between the two groups, with hotter pre-full moon temperatures as predicted by Sarantos (2010). In addition, the full-year plots show broad agreement with the temperatures he proposes for the liberation processes (2010). Cooler emission is in the 1200 K region (PSD), while the hottest is on the order of 3000 K (impact vaporization).

The area data is less conclusive. While the full-year plots, January 2014, and some of the other months appear to broadly support the trend of post-full moon increased emission, May 2014 fails to display major differences in area before and after full moon as it does with temperature. Furthermore, April 2014 demonstrates a heavy dependence in area on the crater used to aim the telescope, indicating a latitudinal dependency in liberation rates as has been previously proposed (Sarantos 2010). A more extensive treatment than that presented here will be required to transform the area data to absolute column densities and correlate these with overall potassium liberation rates.

Conclusion

Spectral line widths of potassium observations were converted to direct temperature measurements of the lunar potassium exosphere. These data appear to confirm a proposed priming of the lunar regolith by plasma sheet ion bombardment during full moon, leading to cooler, increased emission in the days afterward. Spectral line areas have been amassed as well, and while they broadly seem to support the proposed priming, they are affected by atmospheric extinction and demonstrate a heavy latitudinal dependence. More study is needed to investigate these effects before the area data can reliably applied to the question at hand.

References


Figure 4a: K Line Areas, January 2014

Figure 5a: K Line Areas, February 2014

Figure 6a: K Line Areas, April 2014

Figure 7a: K Line Areas, May 2014

Figure 4b: K Line Widths (FWHM), January 2014

Figure 5b: K Line Widths (FWHM), February 2014

Figure 6b: K Line Widths (FWHM), April 2014

Figure 7b: K Line Widths (FWHM), May 2014
BLENHEIM: A BATTLEFIELD

Blenheim was not the common house of any common lord – its unique history, sustained in its numerous unique architectural elements, begins on a battlefield in June, 1704. The following year, the structure commemorated for that battle becomes the field of battle, where ideas of distinct personalities surrounding its construction clash.

The Duke of Marlborough led forces to victory on the banks of the Danube River, defeating the French King Louis XIV’s twenty-eight regiments under Marechal Tallard. Back in London, the English Queen Anne and her close acquaintance, the Duchess of Marlborough, celebrated his victory four weeks later with a service at St. Paul’s Cathedral. This was not to be the end of the celebrations, however; this was only the prelude to a drama revolving around the construction of Blenheim Palace that began with the Queen’s award of the 15,000 acre royal manor of Woodstock in Oxfordshire.

First and foremost a monument to Queen and nation, Blenheim was to be a military victory’s castle-stronghold; its role as a country house for the Churchill family last on the minds of the Duke and his architect. The 1705 edit issued by the Queen officially commissions the “Castle of Blenheim”; reference to the militaristic strongholds of the medieval nobility. The project, therefore, already had the monumental connotation of a national monument in its early stages. It was not to be a regular home from the outset – neither was it to be a structure like any before. Such a grandiose building, neither for the function of monarch or church, was ever built before. However, Blenheim was not called a palace until the nineteenth century. Nevertheless, it challenged the norm - in its conception, execution, purpose, and function.¹

JOHN VANBURGH AND THE GREAT DRAMA OF BLENHEIM

The plans for the “castle” as set forward by Queen Anne would be drawn up by Compter of the Works, John Vanbrugh, and his assistant, Nicolas Hawksmoor. Vanbrugh possessed great talent in many areas and he accomplished much in his life. He studied architecture in France, spent ten years in the military and made a career out of playwriting. He composed several of the most recognizably British comedies of the period. Ultimately, his involvement in the theatre would lead him to construct an actual theater, the Queen’s Theatre in Haymarket, and furthermore, impress contemporaries and historians with perhaps a greater talent still: the design of monumental buildings.²

How ironic, then, as a writer of comedic drama that he himself be a main protagonist, as the leading architect and surveyor, in the Blenheim tale – in itself more tragedy than comedy. Supposedly, Vanbrugh, then working on his first house “Castle Howard,” was selected by the Duke rather than the Queen, giver

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of the reward and the subsequent funds afforded the project by act of Parliament. The Duchess, Sarah Churchill, would say that Vanburgh was the monarch’s choice; however, Vanburgh produced documents—a warrant, in fact, signed by Lord Treasurer Goldolphin—that proved he was appointed at the “request and desire of the Duke” without any reference to Her Majesty the Queen. 3 At the outset, Sarah disagreed with the whole nature of the project. With home, comfort, and economy in mind, she preferred the clean classicism and reliability of Surveyor Christopher Wren, who would later do work for her at their home in London.

Blenheim would take twenty years and over three hundred thousand pounds to build. Its construction was the source of a long, drawn-out conflict between the aforementioned characters of Vanburgh and the Duchess, with the Duke himself off to war and largely irrelevant. The two would scuffle over issues such as the transportation of stone (which came from over twenty different quarries around England, since local stone was not up to par), the disposition of the ancient remains of Woodstock Manor, and the construction of a great, arching bridge so fantastic in the imagination of Vanburgh. Ultimately, the conflict was rooted in their individual interests and ideas, which at heart, could not be reconciled. Vanburgh was a man of theatrical, grandiose concept, who desired to embody the Queen’s warrant to the fullest extent. In a letter he wrote in 1710, he said grandly:

When the Queen had declared she would build a House in Woodstock parke for the Duke of Marlborough, and that she mean’t it in Memory of the Great Services he had done her and her Nation, I found it the Opinion of all people and of all party’s I convers’d with, that altho the building was to be calculat’d for, and Adapted to, a private Habitation, Yet it out at ye same time, to be consider’d as both a Royall and National Monument, and care taken in the design, and the Execution, that is might have ye Qualities proper to such a Monument, Vizt. Beauty, Magnificence, Duration... 4

And yet, Sarah Churchill would stand firm in her concept of the “castle” as a country house—their personal home. As such, she held true to her ideas of simplicity and economy that were in such stark contrast to those of Vanburgh. All the while, the warfare of their personalities was set against the actual warfare waged by Marlborough in the field, as well as, significantly, that conducted in the political sphere. Marlborough was a Tory, like the Queen, and yet their military engagements abroad was of great concern to “High Tories” who resented such intercontinental conflict. Following his victory at Blenheim, the Duke joined forces with moderate Parliamentarians, and the funds for Blenheim were believed to be reliably provided and sustained by the Whig Lord Treasurer Goldolphin. However, a series of defeats in war, the subsequent rise of the Tories headed by antagonist Robert Harley, and the fall of the Duchess from the Queen’s favours eventually led to troubles with funding, a halt in construction, and the Duke and Duchess’ “exile.” They were only to return when Queen Anne died—indeed, the day after!—and upon the ascension of the Hanoverian monarch George I and the rise of the Whigs. Vanburgh was knighted, large debts to contractors forgiven and paid, and work resumed on Blenheim. Although the outlook now appeared bright from here on out, the death of Marlborough was the start of the eventual downslide that

would lead to Vanburgh’s removal from the project. Nevertheless, Sarah continued with the plans and
finished the project, finally completing the erection of a grand palace and park in her husband’s memory.
This unique blend of personal and political developments would make for an interesting blend of
architectural elements, many of which were surprisingly sustained from their initial conception to
completion two decades later. However, I do believe following the death of Marlborough that the nature
of Blenheim changed – conceptually and actually. Sarah had begun to accept the Woodstock estate as a
monument rather than a country house. This was why Vanburgh’s designs continued to be carried out
following the removal of Vanburgh himself. But the monument took on a different meaning for the
Duchess who, without the influence of Vanburgh or even her husband, carried on construction as if it
were instead a monumental mausoleum to honor not the collective nation but a singular man.

A WARRIOR’S CASTLE; A MILITARY MONUMENT

Blenheim was designed to dominate, just as the Duke had done in battle. The structure itself reigns
victorious over a cohesive and controlled landscape – the choreography of the surrounding park had to
blend seamlessly with the style and message of the great stone monument. From the interior, a view of the
formal gardens offers an on-looker that sense of dominance – wild nature yields to man; bushes and
shrubs line up in neat orderly uniforms of geometry and coordination. From the exterior, the castle is the
centerpiece, taking all the surroundings in under its formidable columned arms and omnipresent
windowed gaze.

Like Vanburgh’s first house at Castle Howard, Blenheim is entered through the north side, the façade of
which stretches four hundred eighty feet in length. This weighty front ominously frowns at the viewer;
the density of its bulky columns and pilasters forms a warning grimace rather than an open welcome.
There are no “country houses” to compare with its protruding Corinthian columns and grim recesses of
dark shadow. All around this imposing front, which literally affronts the visitor upon approach, is the
Great Hall, Saloon, and State rooms to the south; the staircase adjacent to both sides of the hall; and the
family room behind the facades of the east sides. The west front housed the Gallery, now the Long
Library. In size and scale, Blenheim dwarfs Castle Howard. Vanburgh’s architectural philosophy was
based on mass – emphasis on the bulk of the structure played into his whole purpose to create a building
of “Beauty, Magnificence, and Duration…”5 Geoffrey Beard calls the structure “semi-Cyclopean” with
those strategically-placed clusters of columns, its projections and recessions that at once assault the senses
with the grandeur and formidable magnificence of the Duke.6 The whole design of the building seems
simultaneously to force the onlooker back and to take him in, draw him to the drama of opulently layered
blocks of stone and the mass assembly of decorations by Grinling Gibbons literally depicting the
theoretical and actual loot of the Duke’s victory over French forces. At the central portico, Minerva,
goddess of war, nobly glares out onto the landscape beyond, two captives at her side. British lions of the
East Court garishly tear at French cocks. The four corners of the structure are towers, each with a
pinnacle bearing the imposing symbol of Marlborough, the coronet, triumphantly defeating French fleur-de-lis below it. All glistened in gilding and color like a row of a thousand formidable armored knights.7
What aristocrat would decorate their home, their country seat meant for leisure, enjoyment, and
entertainment, in such a way? What contemporary cathedral or monarchical pleasure palace would don

5 Vanburgh, 39.
6 Beard, 39.
7 Fowler, 23-76.
such militaristic regalia? Thus was the originality of Blenheim’s architecture: it was neither a country seat, a religious house nor a monarch’s play-place. It served that unique purpose of a monument. It was only suitable that the exterior exhibit such appropriately unique features, which, serving the purpose of monumentality, convey the message of a monumental cause.

Truly, though, the grandeur of size is what impresses the senses and overwhelms a visitor’s sense of scale. House and courtyards taken together, the Duke’s force of stone and gliding covered a total of seven acres.8 Those sharp, sky-piercing towers, grabbing arms, a double-layered pediment-portico, and detailed Corinthian columns with their dramatic, shaded recesses make Blenheim a structure unique even from its predecessor Castle Howard. Similarly, the interior features correspond in magnificent scale and grim grandeur. The Great Hall is a show-place of Marlborough prestige. It is sixty-seven feet high with a magnificent Thornhill painted ceiling depicting the great victory of Marlborough as if he is ascending to the heavens.9 Despite the divine light shining on Thornhill’s painted Duke, there is little actual light: the general atmosphere is one of a “cathedral’s solemnity.” Light trickles in from the clerestory windows atop those horrendously tall walls – in fact, they are the only windows in the whole room. Furthermore, Vanbrugh chose to emphasize the sharp geometry of gargantuan masonry constantly surrounding you; dwarfing you; grimacing down at you with the gaze of an unsympathetic warrior-legend. These were just the elements that made Blenheim the “castle” commissioned by the Queen; the status symbol desired by Vanbrugh and the Duke of Marlborough; and cold rigidity – that came at quite an outrageous expense – abhorred by the Duchess, who could only dream of comfortable elegance typical of a country house of the era.

Her desires were moderately appeased with the “Bow-Window Room,” where soft shapes, soft drapery, and Corinthian pillars of wood carved again by ever-masterful Gibbons took precedence. This particular room also emphasizes the need for the outdoors to blend seamlessly with what was indoors. The bow-window, specifically requested by Sarah Churchill, gazed out over a garden of feminine roses – the only one like it on the grand expanses of order and control. The remaining grounds, designed by royal gardener Henry Wise, stood under the firm command of the house. Even the bridge designed by Vanbrugh (highly contested territory between the architect and the Duchess), arched over three distinct strictly-controlled canals. Only later was it made into a lake for the appearance of natural elegance.10

CONCLUSION

Blenheim seems a place designed for the celebratory processions of ancient Rome. Visitors must march, as victorious soldiers once did, down the long lane of the entrance, passing columns and arches in the mold of those honoring Trajan and Titus. The end of the journey, the pinnacle of this triumphant procession, is Blenheim itself, where one may stand before it like a worshipper before the temple of gods. The “palace” is massive in its sheer size, its architectural opulence, and its collection of sculpted cannonballs and guns all heaped like the booty of the defeated offered by the victorious. Even the stolen bust of King Louis stares down like a severed head on a pike - he ominously warns of the power of his

8 Fowler, 18.
9 David Green, Blenheim Palace, 19-20.
captor—who for all this splendor must not be a mere man but a god! Vanburgh himself declared that his conception of Blenheim was as a structure “worthy of a Roman conqueror.”

Often Blenheim Palace is declared the pinnacle of the English Baroque movement in architecture. Is this true due to its unique status more as a monument to monarch, nation and “conqueror” than a simplistic country house of the Duchess’ dreams? In my opinion, the structure’s architectural elements surely make it more literally a castle that the Queen warranted rather than a nod to the Baroque neoclassical grace of Wren that the Duchess herself had preferred. Its impression of military might makes it of a completely different mold than not only country houses, but other monumental structures like St. Paul’s Cathedral. As a result of its architectural uniqueness, it is really not representative of its times in stylistic terms. Instead, it becomes a symbol unique to its circumstance. Though it was first conceptualized to honor royal and national interests—the victorious Duke of Marlborough being simply the successful expression of those interests—it takes on a more personal quality, not in the Duchess’ sense of homeliness, but rather as a memorial to a single man of the military. In this, it stands out from its era—a unique icon of a unique man and moment in time but not the time itself.

Selected Bibliography


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11 Vanburgh, 48.
Geopolitical Exigencies and the Foundation of Israel-Palestine

Although the Arab-Israeli Conflict is a major issue in today's world, its origins are rooted in political decisions made over a century ago by foreign powers. In order to understand the contemporary complexities of the crisis, it is necessary to delve into the past and to study the historical underpinnings that laid the groundwork for the current political situation. I contend that the Israeli-Palestinian Conflict can be traced back to mutually-exclusive British promises made to the Arab and Jewish people during one of the most horrific wars mankind has ever seen – World War I.

Prior to the Great War, the lands that comprise modern-day Israel and Palestine were under the control of the Ottoman Empire. Once a global power, the Ottoman Empire had experienced a phase of steady decline during the 17th, 18th, and 19th centuries. At the onset of the First World War in 1914, the Turkish government was teetering on collapse. With Ottoman entry into World War I on the side of the Central Powers (along with Germany, the Kingdom of Bulgaria, and Austria-Hungary) the Ottoman Empire’s territorial integrity was at risk if she and her allies lost the war. Befitting the imperialistic models of warfare that has characterized modern-European history, the Ottoman Empire would be carved up by the Allied Powers (France, Great Britain, and Russia).

Indeed, the fragmentation of the Middle East was brokered between Great Britain, France, and the Russian Empire. The Sykes-Picot Agreement, outlined in 1915, and ratified in 1916, was a secret treaty between the Allied Powers that outlined their territorial spoils in the Middle East. After the Central Powers were defeated, the city of Constantinople would be given to Russia, Syria and Lebanon to France, and Iraq and Jordan to Great Britain. Palestine was to be placed under international status and conferred upon after the end of hostilities.

Despite this planned fragmentation of the former Ottoman Empire, Great Britain also entered into correspondence with Sharif Hussein, a key leader in the Arab world, with the goal of instigating an Arab revolt in Ottoman-controlled lands in order to tie down Turkish troops. In July 1915, the British High Commissioner in Cairo, Henry McMahon, entered into correspondence with Hussein. Much of the
exact language is (intentionally) vague, but it is clear that McMahon assured Hussein that in exchange for instigating an Arab revolt in Ottoman lands, the British would ensure the independence of the Arab people after the war and establish a Caliphate with Hussein as the leader. Juridical rhetoric can attempt to rationalize these dual claims, but common sense dictates that the promises made to Sharif Hussein were utterly incompatible with the Sykes-Picot Agreement as well as the ensuing Balfour Declaration.¹

The strategic implications for Great Britain's treachery were clear: the Suez Canal played a major role as a lifeline for the British Empire as it connected the mainland to the British Raj. More than just "British India," the Raj covered the entire Indian subcontinent and comprised modern-day India, Pakistan, Myanmar, Bangladesh, and Singapore. If the Ottomans took control of the Suez Canal, this vital lifeline of troops, supplies, and communication would be cut off. With the British mainland already on the brink of starvation due to Germany's successful U-Boat tactics, and her troops tied down in the Western Front, securing access through the Suez Canal was of utmost importance.

The Balfour Declaration was another key statement that rattled the status quo in the Middle East by promising a Jewish homeland in Palestine. The aspirations of the Jewish people for their own state go back millennia, when in 135 CE, the Jewish people were expelled from Judea by the Roman Emperor Hadrian. Nevertheless, modern Zionism is rooted in the mid-19th century in response to increasingly Anti-Semitic fervor and government-orchestrated pogroms in Eastern Europe. In 1897, Theodor Herzl founded the World Zionist Organization, whose goal was "the creation of a home for the Jewish people in Palestine to be secured by public law."²

Chaim Weizmann, another key Zionist, played a critical role in the creation of the Jewish homeland in Palestine. With his charisma, political connections, and

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pouvoir de l’argent, he was able to convince prominent British politicians that the creation of a Jewish state in Palestine was in their geopolitical interests. Along with Lord Rothschild’s influence, several versions of the Balfour Declaration were drafted, with the final edict published on November 2, 1917 stating:

"His Majesty’s Government view with favour the establishment in Palestine of a national home for the Jewish people, and will use its best endeavors to facilitate the achievement of this object, it being clearly understood that nothing shall be done which may prejudice the civil and religious rights of existing non-Jewish communities in Palestine, or the rights and political status enjoyed by Jews in any other country."

With this document, the fate of the Jewish people was altered forever. But why did the British Empire decide to support Zionism? In 1917, Tsarist Russia fell to the Bolshevik Revolution. The strategic importance of this political development to the Allied Powers cannot be overemphasized; it seemed likely that Russia would withdraw from the conflict, allowing the German Empire to concentrate its forces exclusively on the Western Front. Approximately one million battle-hardened German soldiers would pour into Western Europe, posing a serious risk to the Allied war effort. Given this context, it’s clear why the newly elected British Prime Minister, David Lloyd George, sought the support of the Jewish people in order to influence political decision-making at home.

Not only did Jews have political clout among the nascent Bolshevik government in Russia, but they were also perceived as having significant political influence in the United States (who at this point had not yet entered the war). By supporting Zionist aspirations for a sovereign nation, there was a possibility that Great Britain could reverse Russia’s withdrawal from the war while nudging the United States into entering. Though it may have been an expedient decision, British Prime Minister, Lloyd George, felt compelled to take it.

Whatever political clout the Jewish Bolshevik leaders may have had in the fledgling Russian state, she ended up withdrawing from the conflict. Nevertheless,

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the United States did enter. There are many reasons for this, including Germany's unrestricted use of submarine warfare (most notably the sinking of the Lusitania) the discovery of the Zimmerman Telegram, and potential Jewish influence on U.S. President Woodrow Wilson. It is impossible to delve into Wilson's mind, but Jewish influence and opinion, as well as the promises of a Jewish state (which neatly coincided with Wilson's “Fourteen Points”) may have influenced the President's decision.

It's easy for modern readers to criticize the British Empire for their expedient actions and mutually exclusive promises to both the Arab and Jewish peoples. It is not at all my intention to excuse the British government's actions, but it would be helpful to look at this situation through their perspective. World War I was one of the most devastating international conflicts in world history. In 1916, the British Empire was effectively bankrolling the Allied Powers, spending approximately $75 million USD a week on their war efforts (1916 currency, not adjusted for inflation). When Imperial Germany instituted unrestricted submarine warfare in 1917, the tactics were so effective that Great Britain's civilian population was on the brink of starvation.4

A whole generation of men perished in the face of modern warfare: dismal trenches, poisonous gas attacks, walls of barbed wire, never-ending barrages of artillery explosions, and destruction everywhere. Those that did not die directly in combat often returned home maimed or with severe "shellshock" (Post Traumatic Stress Disorder). When faced with such horrendous exigencies, it's difficult to imagine elected officials not doing everything in their power to protect their population; not doing so would be derelict in their duties. Unfortunately, British decisions had unintended consequences that have laid the groundwork for at least a century of conflict and turmoil, with no conceivable end in sight.

In recent years, researchers and educators alike have become increasingly interested in the role that parents and caregivers play in their children’s education. Collectively, the research in this area suggests that family involvement at school and home is associated with an array of positive outcomes for children. For example, children whose parents and caregivers are actively involved in their schooling tend to miss fewer days of school (Sheldon & Epstein 2004), behave better in the school setting (Harris & Goodall, 2008), and develop better social skills (El Nokali, Bachman, & Votruba-Drzal, 2010) than children whose parents and caregivers are less involved (Kinne, 2013). While this research delineates the vast benefits of parental engagement for children, it neglects the possibility that there are important parental outcomes associated with school engagement, such as those related to subjective well-being and parental self-efficacy.

Building off of the social identity framework, I argue throughout this paper that parents and caregivers who come to identify with their children’s school stand to benefit in terms of their own psychological well-being. Parents will only come to identify with a particular school community under the proper conditions, however. First of all, parents must engage, or involve themselves in the activities of the school, as social interaction precedes social identification (Sherif, 1956; Drury & Reicher, 2005; van Zomeren, Leach, & Spears, 2010). Second, parents and caregivers must view the school climate as equitable, perceiving that within the school setting, they are respected and heard, as perceptions of “procedural justice” facilitate group identification, as well (Blader & Tyler, 2009; Tyler & Blader, 2003).

While the focus of this paper is on parental outcomes, it is to be understood that parental well-being is (directly and indirectly) associated with positive developmental outcomes for the child, as well (Ardelt & Eccles, 2001; Coleman & Karraker, 2003; Gross & Tucker, 1994). I begin this section with an introduction to social identity theory and a summary of the construct of social identity as it relates to individual well-being. I then proceed to describe two theories of social identity formation (i.e., how people first come to identify with social groups). Finally, I discuss the implications for social identity theory and these related identity formation models as they relate to the school context, before introducing the studies at hand and their hypotheses.

**Social Identification and The “Social Cure”**

Central to the social identity framework is the idea that individuals can define themselves in terms of both personal and social identities. For example, when introducing herself at a job interview, an applicant may explain that she is a conscientious social worker who is passionate
about her career. This statement refers to the idiosyncratic aspects of an individual’s character (i.e., her diligence and passion for her work), and thus, represents a description of the applicant’s personal identity. This same woman may then elaborate, explaining that the reason why she is so passionate about social work is because as a Senegalese woman who migrated to the United States as a child, she feels closely tied to ethnic minority and immigrant groups, and wants to help meet their needs via social work. In this portion of the interview, the applicant is speaking about herself in terms of her social identity rather than her personal identity, because she is referring to her group memberships (i.e., ethnic minority group, immigrant group) and how they affect her life.

This anecdote provides a compelling example of how seemingly broad social identities can, and do, affect people’s lives at a personal level. The job applicant explains that she is drawn to social work because of her various group memberships; she identifies strongly with other members of ethnic minority groups (perhaps Black Americans, more specifically) and immigrant groups (perhaps Senegalese immigrants in specific, or perhaps immigrants in general), and these group memberships have influenced her educational path and choice of career. This notion that social identity intersects with personal identity in important ways is integral to the present research.

Essentially, there are three major tenets of social identity theory, the first of which simply states that people are motivated to maintain a positive self-concept, or to think of themselves positively. The second tenet elaborates upon the first, and posits that people are able to maintain a positive self-concept through identification with social groups. (The third tenet, which concerns inter-group comparisons, is discussed within this paper’s Study 2 results section.) There exists a wealth of empirical research in support of the theory’s second tenet: social identification is associated with positive outcomes for the individual. The construct of social identification has been linked to high self-esteem (Bettencourt & Dorr, 1997; Haslam & Reicher, 2006), the provision and receipt of positive social support (Cruwys, S. Haslam, Dingle, C. Haslam, & Jetten, 2014, and decreased levels of clinical depression, suicide (Cruwys et al., 2014); Hawton, Harriss, Hodder, Simkin, & Gunnell, 2001), anxiety, and aggression (Bizumic, Reynolds, Turner, Bromhead, & Subasic, 2009). In fact, an edited volume was recently released on the impact of social identification on psychological well-being and physical health; the authors referred to this phenomenon as the “social cure” (Jetten, C. Haslam, A. Haslam, & S. Alexander, 2011).
Whereas research on the “social cure” has largely focused on clinical outcomes such as depression, anxiety, and self-esteem, self-efficacy is an additional outcome that may be associated with social identification. Broadly, self-efficacy can be defined as one’s beliefs that his or her actions will produce the intended outcomes (Bandura, 1997). According to Bandura’s social-cognitive theory, people who believe they will be successful at a task are more likely to engage in behaviors that allow for actual success on said task (Bandura, 1997). An idea central to the current work is that the support and resources associated with group membership may actually serve to engender the belief that success is possible among individual group members.

More recently from the work on self-efficacy has emerged the narrower construct of parental self-efficacy, or parents’ beliefs in their ability to influence their child and their child’s environment in a way that ensures healthy development and success (Ardelt & Eccles, 2001; Jones & Prinz, 2005). A 2005 meta-analysis found that parental self-efficacy was positively associated with parental competence and to a lesser extent, with parental psychological functioning (Jones & Prinz, 2005). Another study linked parental self-efficacy to children’s socioemotional adjustment and academic achievement through positive parenting behaviors (Shumow & Lomax, 2002). Thus, the outcome variable cluster within the current studies is comprised of more traditional well-being measures (i.e., positive affect, self-esteem, etc.), in addition to personal and parental self-efficacy. Parental self-efficacy, however, is the construct of more interest to me, because within the school context, it follows that parental identity would be the more salient identity.

Social Identity Formation

The previously discussed literature has largely focused on the psychological benefits associated with group identification, but what are the antecedents of social identification? Put differently, how do individuals actually come to identify with social groups, and for what reasons? Before presenting the antecedents of group identification, however, it is critical to clarify the distinction between objective and subjective group membership. Whereas a person may belong to a particular group, this objective group membership does not necessarily mean that the individual identifies with the group in question (i.e., subjective group membership).

For example, the social worker previously discussed is objectively considered Black in the United States simply because of the color of her skin. In her particular case, she also happens to subjectively identify with the group that is Black Americans because, to name a few reasons, she
is happy to be Black, she feels committed to other Black people, and being Black is an important part of how she views herself (Leach et al., 2008). In social psychology, these separate, yet related components of identity are referred to as satisfaction, solidarity, and centrality, respectively (according to the Multicomponent Model of In-Group Identification; see Leach et al., 2008). People who objectively belong to a particular social group come to subjectively identify with that group over time and for a variety of reasons. Once they feel this subjective identification with a particular group, they come to experience these variables such as identity satisfaction, solidarity, and centrality, and it is only at this point that their identification with the group will wield any influence over them on an individual level (Leach et al., 2008; Turner & Oakes, 1997). The question, then, remains how, precisely, individuals come to subjectively identify with a particular social group.

**Social interaction as a means of identity formation.** It has traditionally been argued that social interaction and the pursuit and realization of common goals can lead to group identity formation. In Sherif’s seminal “Robbers’ Cave” experiment (1956), for example, young boys at a summer camp came to identify with their group(s) only after having worked alongside other group members in pursuit of common goals. During the initial phases of the experiment, the boys were split into two distinct groups, each of which adopted a group name and then, an identity after competing in a series of sporting events against the opposing group. By the end of camp, however, the boys had come to identify as one, superordinate group, viewing their once enemies as comrades. This social identification came about as a result of all of the boys working together to achieve a common goal: they had fixed the camp’s water source and raised enough money so that the entire camp could afford to watch a movie.

**END OF WORK SAMPLE**