Occam's Razor

Volume 6 (2016) Article 1

2016

Occam's Razor Vol. 6 - Full (2016)

OCCAM'S RAZOR

Occam's Razor *is an annual publication comprised of exceptional student academic writing from the departments and colleges of Western Washington University.*

Author biographies are available on our website, wp.wwu.edu/occamsrazor

This issue of OR *was printed on Mohawk Everyday Digital text 70# and cover 80#, using the fonts Akzidenz Grotesk and Adobe Caslon.*

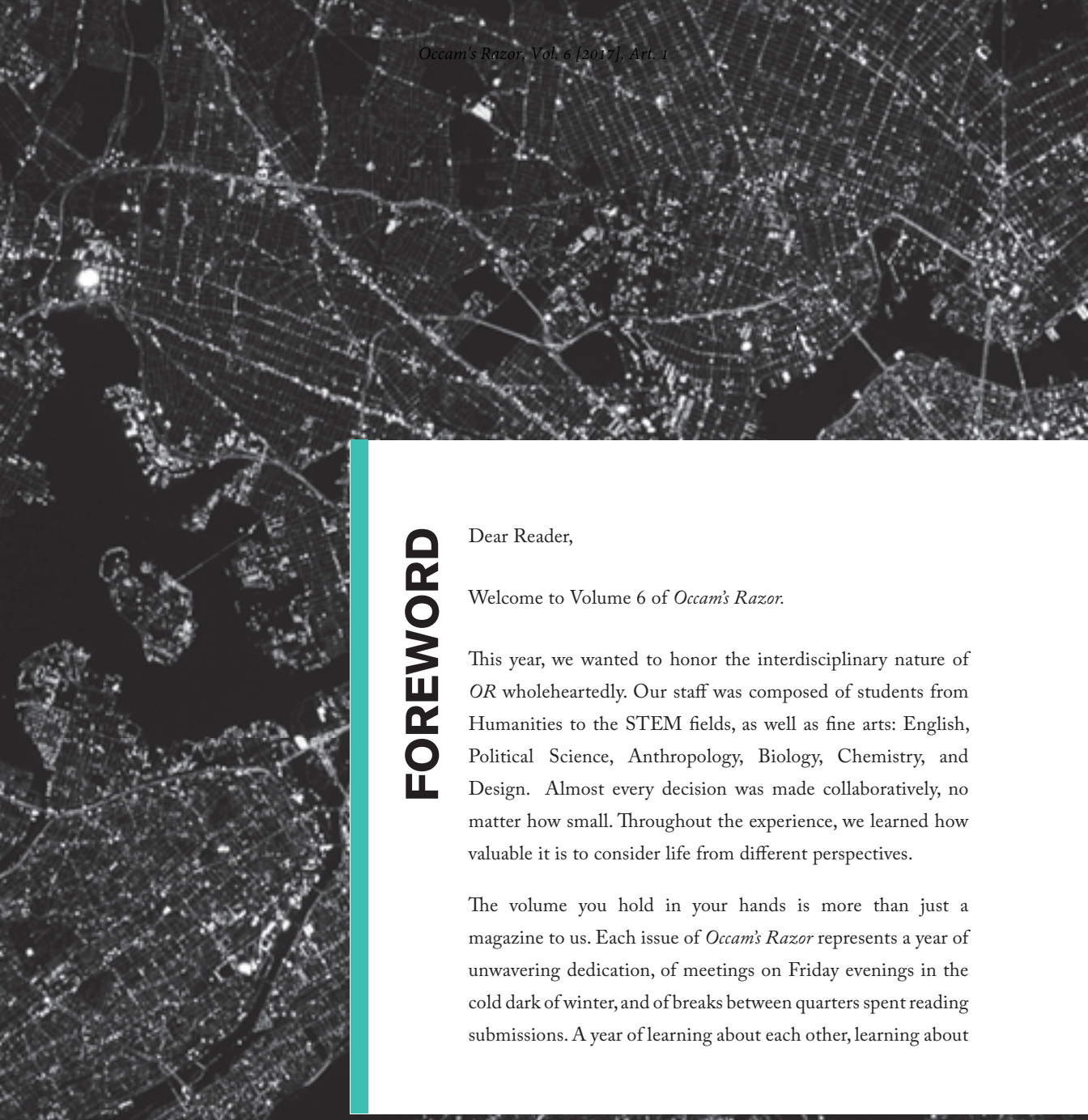
Ockham, William. c. 1495. Quaestiones et decisiones in quattuor libros Sententiarum Petri Lombardi. Ed. Lugd. i, dist. 27, qu. 2, K.

Occam's Razor /äkəmzˈrāzər/

Often called *lex parsimoniae*, or law of parsimony
"Numquam ponenda est pluralitas sine necessitate"
[Plurality must never be posited without necessity]

— *William of Ockham*

Published by Western CEDAR, 2012



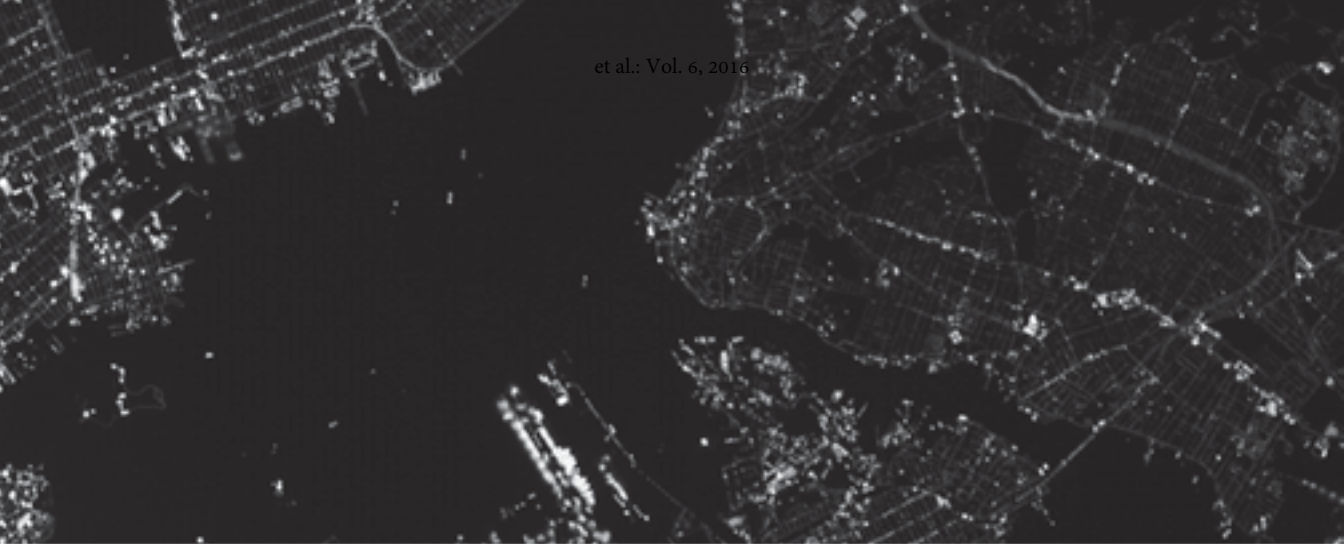
FOREWORD

Dear Reader,

Welcome to Volume 6 of *Occam's Razor.*

This year, we wanted to honor the interdisciplinary nature of *OR* wholeheartedly. Our staff was composed of students from Humanities to the STEM fields, as well as fine arts: English, Political Science, Anthropology, Biology, Chemistry, and Design. Almost every decision was made collaboratively, no matter how small. Throughout the experience, we learned how valuable it is to consider life from different perspectives.

The volume you hold in your hands is more than just a magazine to us. Each issue of *Occam's Razor* represents a year of unwavering dedication, of meetings on Friday evenings in the cold dark of winter, and of breaks between quarters spent reading submissions. A year of learning about each other, learning about



ourselves, and gaining knowledge both within and outside of our own areas of study through the words of our peers. Our staff has cherished every moment!

The essays that appear in this volume represent the finest work of Western's active minds; authors who are passionate and dedicated to the attainment and sharing of knowledge. Even our brief encounters with these individuals were enriching. For that, we give our heartfelt thanks and admiration.

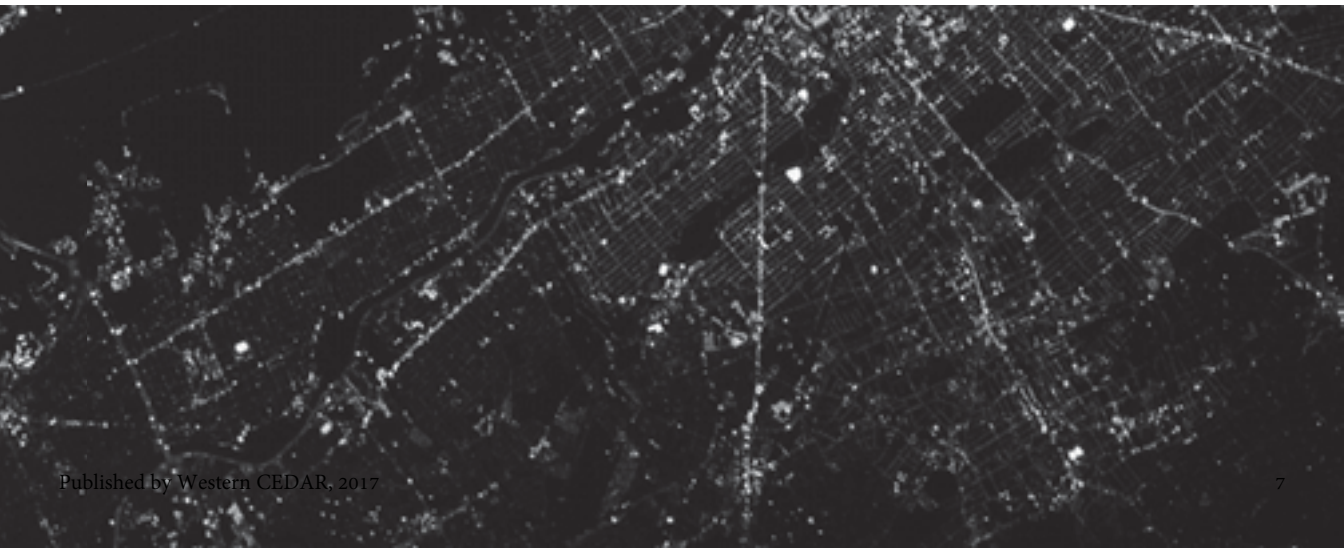
We also give thanks to our faculty advisor, Christopher Patton, for his guidance and support, and for always finding the time for us when we needed advice. We are thankful *OR* has such an invested mentor.

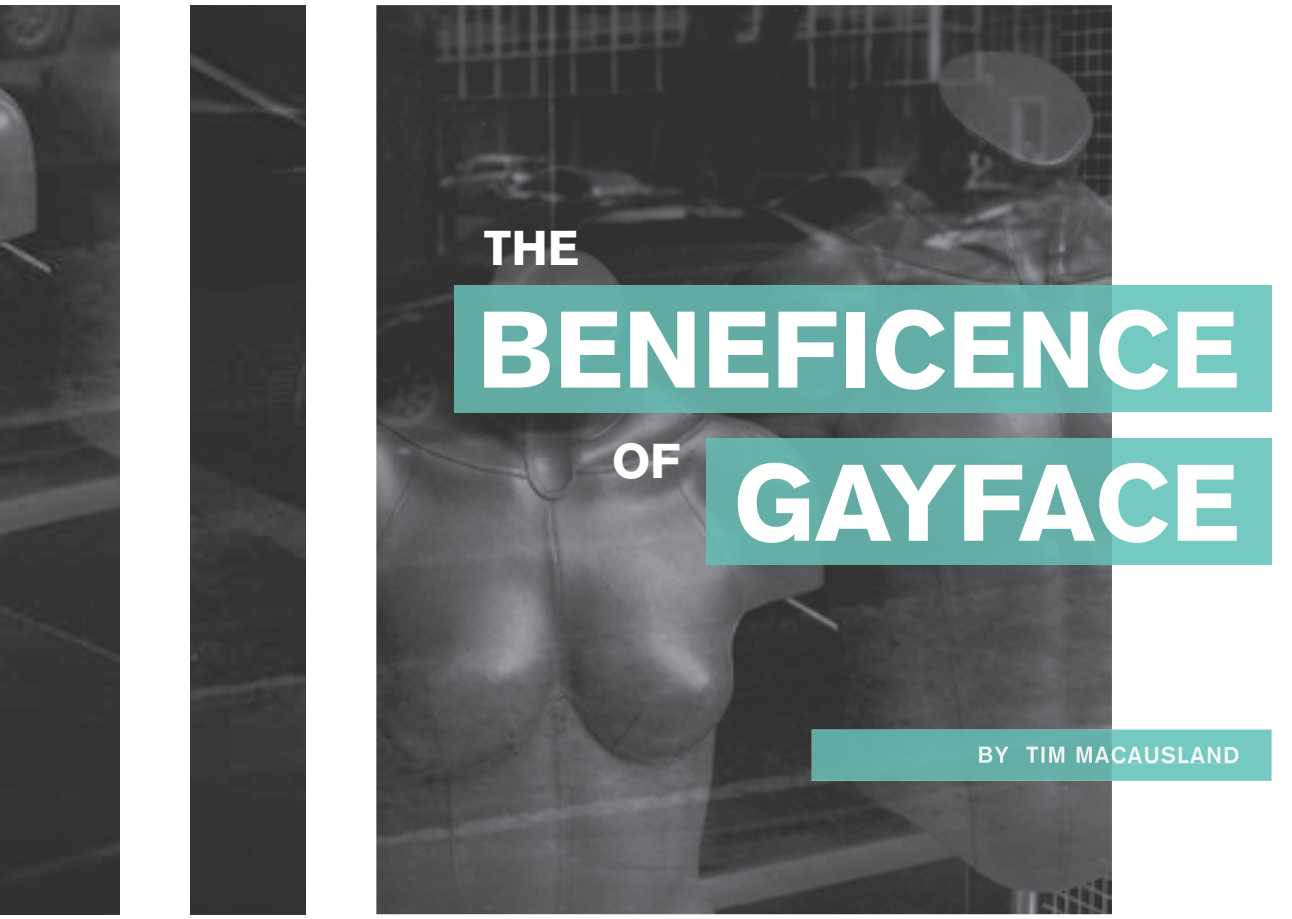
Last—but not least—we thank you, the reader, for picking up this volume. Without your interest, publishing *Occam's Razor* would not be possible.

We hope you enjoy it!

Sincerely,

The *OR* Staff





THE BENEFICENCE OF GAYFACE

BY TIM MACAUSLAND

In 2009, veteran funny man Jim Carrey, best known for his zany and nearly cartoonish live-action performances—perhaps none more literally than in the 1994 film The Mask (Russell, 1994)—stretched his comedic boundaries with his portrayal of real-life con artist Steven Jay Russell in the film *I Love You Phillip Morris* (Requa, Ficarra, 2009). Despite earning critical success and some of Carrey's highest praises of his career, it made many filmgoers who saw it turn their heads in wonder, though not for Carrey's distinct yet animated leading performance. What gained the attention of many critics were his scenes with co-star Ewan McGregor, who played the eponymous character and the target of Russell's affections (Requa, Ficarra, 2009). Audiences were not caught off guard by the fact that the characters were gay; homosexuality had already broken through to the mainstream within the previous decade with films like *American Beauty* and *Rent*. It was, rather, that the actors themselves were not gay. However, they never let it show or undermine the believability of the roles they played. As expected, the stars received much of the acclaim, but the film does represent a peculiar quandary in the ethical value of straight actors in gay roles. This practice is known as gayface, which, though commonly used to encompass all queer identities, also has counterparts that are more specific in transsexuality. Nonetheless, despite the apprehension they elicit, performances like these exemplify the need to tolerate and encourage gayface for the prospect of sexual equality.

Though perhaps not as well known or culturally notorious as its racial counterpart, a rudimentary understanding of gayface can be comprehended

through the sordid history and evolution of blackface. Once a ubiquitous and racially charged practice in film and theater leading up to the U.S. Civil Rights Movement, blackface consisted of typically Caucasian actors applying heavy makeup, such as shoe polish, to present themselves as someone of African American descent, ordinarily to proliferate harmful stereotypes to humorous or hostile effect. It was perhaps most notoriously observed in the film *The Birth of a Nation*, which depicted African Americans as sexual deviants and Ku Klux Klan members as virtuous people (Griffith, 1915). Blackface has been ostracized in contemporary media and only ever appears in more self-aware, satirical commentary of itself. Such examples include Robert Downey Jr.'s Oscar-nominated role in *Tropic Thunder*, where he appears as an overly devoted Australian method actor controversially cast outside his race (Stiller, 2008). As Michael Rogin details in his book *Blackface, White Noise: Jewish Immigrants in the Hollywood Melting Pot*, "Blackface is a form of cross-dressing, in which one puts on the insignias of a sex, class, or face that stands in binary opposition to one's own" (30). Much in the same way blackface aimed to single out African American culture as something perverted in relation to what was widely considered as the "ideal" American culture, namely a white one, gayface has its own—albeit subtler—origins in cinema.

Indeed, blatant homosexuality depicted in American films was largely unheard of before the Motion Picture Production Code (or Hays Code) citation prohibited it from 1930 to 1968, which cited the behavior as "indecent" for public audiences to spectate. The allusion to homosexuality was permitted, as the director could slip it past the censors by keeping the character's sexual orientation unconfirmed and incidental to the plot. Out of these stipulations, the "sissy" archetype emerged, most prominently in the 1930s, and with it came some of the earliest instances of gayface (Benshoff 14). As opposed to blackface, early depictions of gayface in the form of the sissy were not quite so malign or openly degrading. Fitting in with Hollywood's earlier propensity for more lighthearted fare, wherein comedies typically highlighted the heterosexual ideal directly, the sissy represented a man whose effeminacy served to fulfill a lovably pitiful character. Such a character acted as a foil to the more than likely straight romance that drives the narrative; his lack of masculinity accentuates that of the male protagonist (Benshoff 16). This is the case with *The Dickson Experimental Sound Film*, a seventeen-second video featuring two men dancing to the melody of a violinist, which many consider the first instance of suggested homosexuality in a moving picture (Dickson, 1895). The sissy's sexuality was superficially metrosexual at best, and seldom did his mannerisms or disposition elicit strong disapproval in audiences. He behaved as a whimsical fool or a persnickety stooge. His latent homosexuality registers in the viewer's mind as no more than an unconscious understanding, or at the very least a doubt, that whatever sexuality he embodies is of little consequence to the viewing experience.

OUT OF THESE STIPULATIONS, THE "SISSY" ARCHETYPE EMERGED, MOST PROMINENTLY IN THE 1930s

As the decades have waned on and the forbidding of gay characters—previously under the classification of "sexual perverts" by the Hays Code—has been lifted to the point of their mainstream status, there has been a dramatic shift since the days of the sissy. Well-written queer parts have transformed the role of the homosexual from something ridiculed or pitied, to something that is highly sought after due to their newfound emotional pull with audiences, thereby making them critically lucrative. This gives the actor a suitable range to spread their talents beyond their own sexuality—assuming they are, in fact, straight actors employing gayface—and elicit praise otherwise not accessible with the oversaturation of straightness. Of course, Hollywood has always recognized great transformation, such as Christian Bale's weight loss for *The Fighter* or weight gain for *American Hustle*. Bale was nominated for an Oscar for his work on both, and was awarded Best Supporting Actor for the former ("Christian Bale Biography"). In a similar manner, queer identities suffused with poignancy or reflective of controversy are commended, such as Hilary Swank's performance in *Boys Don't Cry* or Heath Ledger's and Jake Gyllenhaal's in *Brokeback Mountain*. For many actors seeking total character engrossment, it is not necessarily the tendentiousness of a queer role that allures them. In an interview regarding his film *Philadelphia* (Demme, 1993), wherein he portrayed a homosexual man afflicted with AIDS, Tom Hanks expressed,

> People are saying that I was bold to do this, that it was a courageous choice. I don't see it. It's bold for me to do what? To play a man who goes to sleep in Antonio Banderas's arms every night? Who has sexual intercourse with him somehow? Is that what's bold? As a society we should be beyond that. (Hanks)

Nevertheless, with the advent of known straight actors tackling roles outside their sexualities, so too has arisen a lesser-known controversy around the morality of gayface.

Similar to how blackface is condemned for its stark and unflattering representations of African Americans, gayface has received its share of criticism over the years, though certainly not enough to make much of a dent in its prevalence, if at all. Hollywood has matured from the concept of the sissy and other more offensive archetypes with its ever-expanding liberalism, but there still exist movies that rely on their homophobia within the narrative. A blatant example of this is *I Now Pronounce You Chuck and Larry,* a film targeted specifically to straight men. This gives cause for concern for gay moviegoers, and understandably stimulates a desire to ensure that films advocate

homosexual relationships rather than making profit at their expense by putting them in a harsh light. Admittedly, however, Adam Sandler and Kevin James do not fall under the category of gayface for their collaboration. They are both straight actors that portray homophobic straight men portraying gay men, but they expect the audience to chuckle every time the famously gay-for-pay Nick Swardson prances about in a butterfly costume with a preponderance of glitter speckled upon his bare chest (Dugan, 2007). Though by the end of the film an apparent pro-gay and overall accepting message is conveyed, some pictures take a more negative approach. Take for instance *The Silence of the Lambs*, a Best Picture winner that is often considered one of the finest films of recent decades (Demme, 1991). However, one cannot help but cringe at heterosexual actor Ted Levine's character Buffalo Bill, a transsexual who also happens to be a maniacal sociopath. His sexual repression "forced" him to slaughter a handful of women to complete his transformation into the female sex by means of a flesh suit made out of their skin (Demme, 1991). Some cite this example as one of the worst offenders for films that perpetuate the trope of having an LGBT character as the villain; his "savage" sexuality becomes the sole origin of such evil and can only be defeated by the straight-laced protagonist. LGBT communities have valid fears when it comes to a straight actor crossing the threshold.

Perhaps the greatest cause for anxiety over the representation of homophobia in film through the execution of gayface stems from the awareness of the total control these straight actors and directors have in the final product of these queer personas. Like any disenfranchised population, queer-identified individuals have absolute reason to be wary of representation by those people who identify with an opposite sexuality, whose bigotry has both disenfranchised and caused acts of hateful violence against them. Technically speaking, any homophobic filmmaker given the authority to depict queer lifestyle in a harsh and degrading light could do so to the same effect as blackface, both in terms of pervasiveness and infamy. Such development could easily and single-handedly define what it means to be queer in the eyes of the viewing public, much in the same way propaganda operates. Thankfully, the LGBT rights movement has made great efforts toward mitigating those fears; it is almost safe to presume that gayface will be used with good intentions, as filmmakers wish to avoid widespread condemnation.

An economic reason for having issue with gayface is the purported displacement of openly gay actors from gay parts. Some feel that such roles should be restricted to those actors who already subscribe to the scripted sexual orientations, thinking they would be

more inclined to fit the part. For instance, Christopher Kelly of *Salon*'s succinct reproof of the film *Behind the Candelabra*, which stars the straight actors Michael Douglas and Matt Damon as the pianist Liberace and his gay lover Scott Thorson, (Soderbergh, 2013) asks the reader whether it may be "time to say thanks but no thanks—and demand that gay artists tell these stories instead?" (Kelly). It is admittedly an admirable proposal, one that caters to the careers of those actors who, more often than not, are playing straight and thus outside their natural disposition; however, there are some repercussions that follow such a proposition.

For starters, considering the reversal—gay actors and straight roles—would make for quite the inhibiting double standard. The logical question that would arise is whether straight roles would likewise be restricted to straight actors. Granted, queer individuals could hardly damage the reputation of the straight community through their portrayals in quite the same way. However, if society's gradual shift toward sexual equality is wholly realized, such a double standard would come into effect. By no means does such a double standard need to be implemented at the present time. But there may come a day that performances from openly homosexual actors, such as that of Neil Patrick Harris as the womanizing Barney Stinson on the critically acclaimed sitcom *How I Met Your Mother,* or Ellen Page's performance as the straight title character in *Juno,* are publicly disallowed. With the aftermath of this actor-character sexual correspondence, queer actors everywhere—the very ones the proposal would seek to protect—would suddenly find themselves at a loss for work, the bulk of written roles intended to be either heterosexual or undefined. GLAAD reports that only 16.7% of the major film releases in 2014 included queer-identified characters (Wong). No matter the potential shift a progressive society can have on said statistic, it would do more harm than help to limit these actors to queer roles.

One other gripe people have with the actors of gayface themselves is that it can force them outside their sexual comfort zone by having to project intimacy with a partner whom they would not otherwise cozy up to. One would not necessarily have that in mind when watching a film, but it is something that many actors have to overcome, whether they are performing outside their sexuality or not. To frame it within the realm of gayface, one could look at the controversy surrounding the breakout French film *Blue Is the Warmest Color.* Though much of the off-screen tension came by means of Abdellatif Kechiche's questionably opprobrious directing style, stars Léa Seydoux and Adéle Exarchopolous reflected that they were "pushed further than they wanted to go on screen," with Seydoux remarking that she "felt like a prostitute" (Del). It is a problematic barrier, but if disappearing into a role were an easy task, anyone could

be an actor. "Part of the job is making yourself comfortable in situations that are not familiar," an anonymous heterosexual actor reflects in an interview with Nicholas Brown of *The Atlantic*. Indeed, but despite the expectation on the industry, there truly is more of a sociological and psychological reluctance to market one's image as homosexual in nature. In the interview, the anonymous actor goes on to say, "I don't want people to think I'm gay. And I'm even more uncomfortable because that isn't a thought that I want to have" (Brown). On the other hand—that of the viewer apprehending a straight person playing gay—actor Harry Hamlin of *Making Love* perhaps best summarizes the internal process of understanding gayface with his comments in the documentary *The Celluloid Closet*:

> I am sure that inside of me there is the same homophobia that we all share. If I see a guy who is playing a gay role, I'll question it. I'll say, 'Wow, is he gay?' And why I do that, I don't know. But then I'll stop myself and say, 'Hey, that's really ridiculous. You know; you've been there; you've done that.' You know the question is, 'Why do we care?' Who cares?
>
> (Epstein, Friedman, 1995)

The subject both actors touch upon, but do not entirely broach, is one that has been rooted in queer theory for quite some time. This has been defined as the *homosexual panic*: the fear of being gay or being judged as gay (Sedgwick 19).

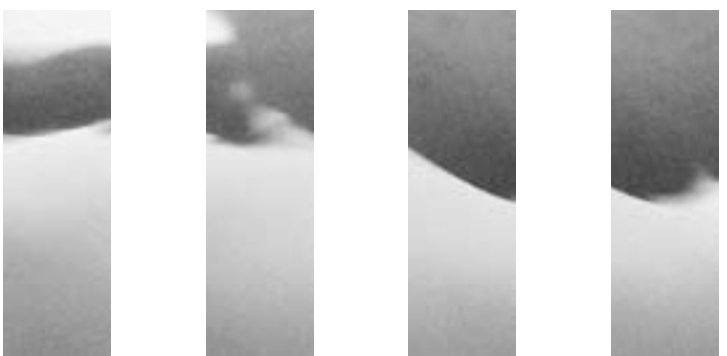
In her book *Epistemology of the Closet*, Eve Kosofsky Sedgwick discusses this homosexual panic in more criminological terms. She writes that it is a "defense strategy," wherein "a person (typically a man) accused of antigay violence implies that his responsibility for the crime was diminished by a pathological psychological condition, perhaps brought on by an unwanted sexual advance from the man whom he then attacked" (Sedgwick 19). Though the judicial claim can still hold some degree of credence in contemporary court (sans in California, which officially debarred the defense in 2014), this still relates to what Brown's anonymous actor outlines: connoted homosexuality pressed onto a straight individual can elicit an averse response by means of their either latent or fully realized homophobia. It would appear that the manifested homophobia with Brown's interviewee and Hamlin also evokes morally contrite responses, as opposed to those who unabashedly employ the gay panic defense. It does bring up an interesting predicament in the practice of gayface, namely that, as the actor detailed, above all else behooves a casting director to hire performers who

HOMOSEXUAL PANIC : THE FEAR OF BEING GAY OR BEING JUDGED AS GAY

are actually capable of quashing their discomfort with any given part.

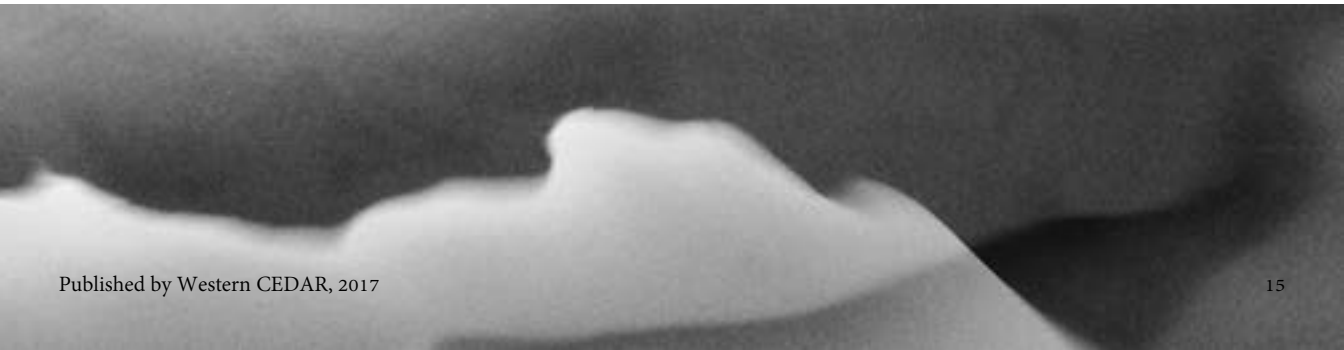
Of course, above all the criticism gayface has generated, there is the quintessential complaint that plainly cites the actor's polar sexuality in relation to the character as a case against its implementation. In short, some feel that representing a human characteristic as culturally delicate as sexuality without the foundational and inherent exposure to it is downright egregious and offensive, much in the same way blackface is viewed. However, the discourse on the matter suddenly becomes blurrier when an individual's supposedly innate sexual binary is removed; when one considers that they are not actually locked into a specified orientation, so to speak. In Judith Butler's *Gender Troubles*, she claims that identity is performative, that "there need not be a 'doer behind the deed,' but that the 'doer' is variably constructed in and through the deed" (195). In application to one's sexuality, one could then posit that it is not so much that being inherently heterosexual, homosexual, or any other sexual variation defines one's orientation itself. Rather, it is the act of finding one's sexuality that is ultimately projected. Such would prove to be a solid case for gayface, wherein virtually straight actors must find some justifying, overarching truth that makes it permissible to portray some intrinsic quality that does not necessarily accord to what they have predominately considered themselves to be. Rather than pretending to be gay-for-pay, they are gay to some extent through their real-life actions, despite the fact that they are following a written script.

Though this would not particularly harmonize with how it is typically presented to a viewing public, I personally have had first-hand experience of gayface through this projected sexuality and gender that Butler describes. When I was nineteen, I participated in a stage show at Peninsula College of Peter Shaffer's *Equus*. The plot follows an impressionable, teenage boy named Alan who realizes his sexuality through his pseudo piety and erotic worship of horses, which he believes is embodied by an omnipresent horse god. Much of the material does not shy away from the ineffable, as Alan praises his god by riding his favorite horse, Nugget, bareback, until he reaches his sexual climax. The show ends with Alan's psychiatrist unearthing his repression of blinding six horses with a hoof pick, in an act of defiance to his envious and unmerciful god. As Alan, I found myself utterly aroused by the material and characterizations, despite the fact that I have never humored the sex appeal of a horse, or bestiality, in my life. My projection of Alan's attraction for Nugget, a character portrayed by a very muscular and handsome (straight) man who sported a skintight unitard for the production, is an example of Butler's concept of projected sexuality. In the first scene, Alan and Nugget tenderly



embrace and breathe down one another's necks, gingerly stroking each other's skin in conjunction with the psychiatrist's soliloquy. Now, I, being a straight man outside all thespianism, have never found myself sexually attracted to another man in my life. However, on that stage, as Alan, with the ever-receptive Nugget sending me scores of energy like a good actor should, my own sexuality transcended into something else entirely. Suddenly I was Alan, caressing Nugget and listening to my own heartbeat race. Since that show, I have worked with that same actor on multiple occasions, and have never once rediscovered that intensity I felt onstage. Such is the same with some actors who stumble upon an alternate avenue of their sexuality through a role and ultimately redefine themselves in the process. Now, compare that with a show I did about a year later called *Ondine*, wherein I had to kiss a male actor in drag due to the lack of female cast members to fill the part. The reason nothing resonated with me sexually during that scene is attributed to the levity of the interaction; it was never intended to be taken seriously, and only served as a bit of comedic relief. It was great theater in its own right, but it takes a truly solid immersion into a character to produce a sexuality that is so wholly other. This sort of unrealized, pseudo-sexuality plays into Sedgwick's detailing of what she calls the "universalizing view," which details that such homosexuality is rather something that exists in everyone to varying degrees of materialization (Sedgwick 1).

Oftentimes detractors of gayface focus on the details surrounding the performance, such as the sexuality of the actor, but seldom evaluate the performance itself, the intentions behind it, and the overall effect it has on mainstream audiences. When taking into consideration films that have been lauded not only for their cinematic heft, but also for their overarching progressive themes, one would find that non-queer actors headlined many of these projects with a queer protagonist; again, Tom Hanks in *Philadelphia* serves as a great example. Yes, there are some ostensibly homophobic films that require viewers to tread lightly, à la *I Now Pronounce You Chuck and Larry*, but by and large Hollywood is producing more and more films that appeal to pro-gay audiences. When considering the dispute surrounding gayface, J. Bryan Lowder states, "Part of the gay community's patience with gayface has to do with a kind of representational pragmatism: Many gays are so happy to see a story like Harvey Milk's told at all that they're willing to cede the role to Sean Penn" (Lowder). As well they should be, considering that *Milk* is perhaps the seminal motion picture in support of the gay rights movement. It was a film produced to favorably reflect society's ever-liberalizing stance on sexual equality in the face of unwarranted homophobia (Van Sant, 2008). Though Hanks and

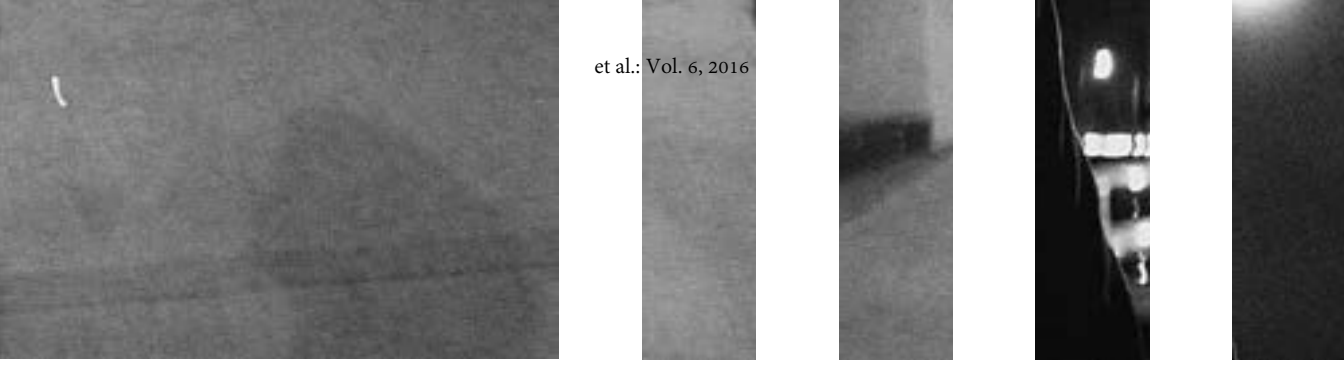


IN ORDER TO PROMOTE A REFLECTIVE SEXUAL BALANCE, AN ACTOR WHO IS ABLE TO FILL A ROLE COMPLETELY AND DO IT JUSTICE SHOULD PLAY THE PART

Penn, both of whom won the Best Actor Oscar for their respective work, are straight men, one should consider the support they gave the gay community by bringing not only homosexuality, but intimate, "alternative" sexual expression into a positive light (Van Sant, 2008).

Of course, though the queer rights movement has been making gradual headway with the advent of the twenty-first century, there still remains a just reason to be wary of surfacing homophobia in public outlets. Needless to say, American culture has not yet achieved sexual equality, and sociological precautions must be made to prevent negative outflux against the movement. However, the prohibition of gayface should not be one of these precautions. It is understandable to be cautious of homophobia leaking out on-screen at highly discernable and ostensibly mainstream rates, but for the most part, the film and television industry has adapted to fit the standards of the contemporary and liberal viewing public, and so projects containing malice against queer identities are rarely green-lighted for production. It is perfectly safe, therefore, for Hollywood to set aside sexual inhibitions, and employ the foresight to not only preview, but also provoke egalitarian endgames in terms of total sexual equality. Indeed, one would presume that in a perfect world, sexuality would be less of a glaring stigma or taboo and more of a triviality, whereby for instance a character's orientation—be it gay, straight, bi, trans, etc.—would be inconsequential not only to most plots, but also to his or her merit by the conclusion of the film's narrative arc. Thus, if a character's sexuality is of little importance in relation to the theme, the same logic should apply regarding the sexuality of the actor playing that character. Rather, in order to promote a reflective sexual balance, an actor who is able to fill a role completely and do it justice should play the part.

A large reason why many actors delay their coming out of the closet until they have established their careers and fan bases is the fear of typecasting; once the cat is out of the bag, they will never again be seriously considered for straight roles. It is indeed a valid fear, one that was perhaps most famously demonstrated by the late Rock Hudson, an iconic heartthrob in mid-twentieth-century cinema. Though he never publicly identified himself as gay, up until his death from AIDS during the epidemic of the 1980s, it has long been recognized within the industry as truth (Benshoff 203). In fact, Hudson was known to employ the use of "female beards" to conceal his homosexuality, including his sole wife Phyllis Gates. One cannot particularly blame Hudson,



who, for the bulk of his career, hinged on his marketability as what a typical male lead was expected to be. In other words, he employed what could be considered as straightface almost exclusively. He even portrayed a straight man pretending to be gay as part of an elaborate ruse to bed Doris Day's character in the romantic comedy *Pillow Talk*, another meta performance with the application of hindsight (Benshoff 93). On a macro level, all of this can argue for the disaffiliation between an actor's private and professional life, in the hopes that the former should have no sway over the latter. Of course, this would devalue the potentially opportunistic decision of a straight actor to play a queer character, in the hopes of critical acclaim; but the choice of an actor-character combination should be made based on how it enhances the film itself, not the possibility of accolades.

This comprehensive argument for gayface does not call for its monopoly. It does not, by any means, suggest that gay actors cannot inhabit gay roles, for that would simply be counterproductive. Fundamentally speaking, it only advocates its continued employment in Hollywood and elsewhere without an inhibiting consciousness, which elicits such meticulous micromanagement of the casting process. Rather, it is more in favor of a laissez-faire approach, one that does not take an actor's sexuality in account at all. In fact, there have already been cases wherein gayface actors have performed opposite actual gay actors with much success; cases wherein the parts fit the actors, rather than the other way around. The television sitcom *Modern Family* is perhaps the most exemplary of these, exhibited by the widely commended onscreen relationship between openly homosexual Jesse Tyler Ferguson and heterosexual Eric Stonestreet as the gay couple Mitch and Cam. Essentially, if there is anything their numerous combined accolades are evidence of, it is that not only can gayface be productive, it can also coexist and thrive off of an authentic queer performance.

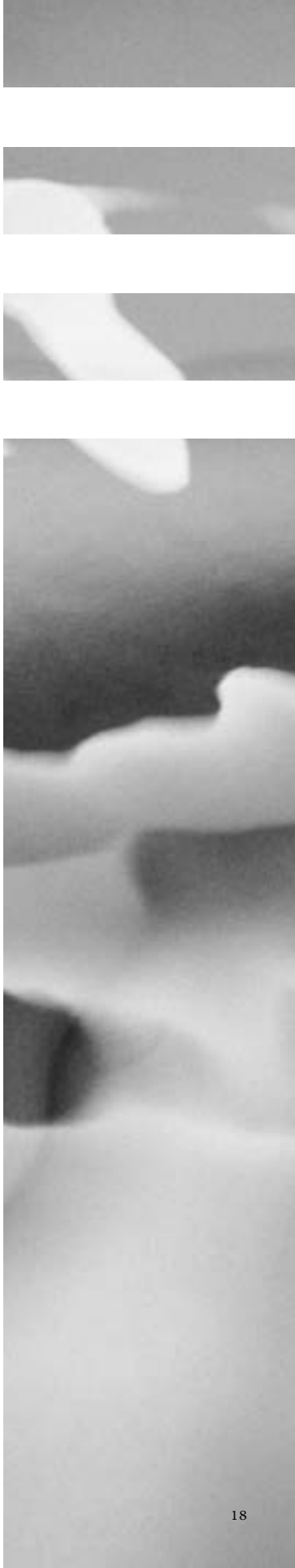
In a pragmatic attempt to further divide the actor from the part by tearing down the fourth wall, what makes the discourse of gayface more intriguing is the role of the camera as the catalyst in the entire discussion. To put it mildly, were cameras not to be rolling, it is doubtful that anyone would care whether the actors interact in such a physical manner. No one would take issue with Jim Carrey and Ewan McGregor actually having sexual intercourse, should they suddenly have the desire to; it is only when it is put to celluloid that their gayness is considered potentially harmful to the

queer community. This highlights the mainstream audience's difficulty with sexual experimentation in reality and on-screen.

Homophobia exists in human society and, like all forms of bigotry; it is not something that is likely to be entirely extinguished. There is no problem with looking back on the grueling and still incomplete road to sexual equality as a helpful reminder, but one must also keep an eye on future dealings; an endgame, if you will. Though gayface is not quite so paramount now, it will be very much so in the generations to come, in order to cement a maximally egalitarian society. At the very least, gayface should be employed if only for the actors themselves, who, like me, will be able to unlock alternate components of themselves through their characters' sexualities. In French philosopher's Michel Foucault's study *The History of Sexuality*, he posits,

> The truth is drawn from pleasure itself, understood as a practice and accumulated as experience; pleasure is not considered in relation to an absolute law of the permitted and the forbidden, nor by reference to a criterion of utility, but first and foremost in relation to itself; it is experienced as pleasure, evaluated in terms of its intensity, its specific quality, its duration, its reverberations in the body and the soul. (57)

Even if gayface actors can reach this summit in their performances, Foucault's argument should be the only rationale necessary to ensure its survival. In fact, gayface, though relevant, ideally should not be a term used often at all. It should be something seldom thought of, more of an afterthought in the grand scheme of the performance. This is how gayface fits into a sexually equitable society: present, but incidental to the overall depiction of human character.



REFERENCES

Behind the Candelabra. Dir. Steven Soderbergh. Perf. Matt Damon, Scott Bakula, and Eric Zuckerman. Jerry Weintraub Productions, 2013. Film.

Benshoff, Harry. *Queer Images: A History of Gay and Lesbian Film in America*. Lanham: Rowman & Littlefield Publishers, Inc., 2005. Google Book Search. Web. 13 May 2015.

Brown, Nicholas. "*Why Do I Still Feel Uncomfortable Playing a Gay Man on TV*?" The Atlantic. Atlantic Media Company, 27 Dec. 2012. Web. 14 Apr. 2015.

Butler, Judith. *Gender Trouble: Feminism and the Subversion of Identity.* New York: Routledge, 1990. Print.

"*Christian Bale Biography.*" IMdB. IMdB, 15 Oct. 2015. Web. 30 May 2015.

Del, Chris. *'Do Great Performance Justify "Gayface"?* - StageBuddy." StageBuddy. N.p., 23 June 2014. Web. 30 May 2015.

Foucault, Michel. *The History of Sexuality*. Vol. I. New York: Vintage, 1978. Print.

Hanks, Tom. "*Playing the Part.*" Interview by Emily Rizzo. Newsweek 14 Feb. 1994: n. pag. Google Groups. Web. 26 May 2015.

I Love You Phillip Morris. Dir. Glenn Ficarra and John Requa. Perf. Jim Carrey, Ewan McGregor, and Leslie Mann. EuropaCorp, 2009. Film.

Kelly, Christopher. "*Are Straight Actors in Gay Roles the New Blackface?*" Salon. N.p., 12 June 2013. Web. 30 May 2015.

Lowder, J. B. "*The Curious Case of Gayface.*" Slate. N.p., 6 June 2013. Web. 14 Apr. 2015.

Milk. Dir. Gus Van Sant. Perf. Sean Penn, Josh Brolin, and Emilie Hirsch. Focus Features, 2008. Film.

Philadelphia. Dir. Jonathan Demme. Perf. Tom Hanks, Denzel Washington, and Roberta Maxwell. TriStar Pictures, 1993. Film.

Rogin, Michael P. *Blackface, White Noise: Jewish Immigrants in the Hollywood Melting Pot*. Berkeley: U of California, 1996. Google Books. Web. 13 May 2015.

Sedgwick, Eve K. *Epistemology of the Closet*. Berkeley: U of California, 1990. Print.

The Birth of a Nation. Dir. D.W. Griffith. Perf. Lillian Gish, Mae Marsh, and Henry B. Walthall. David W. Griffith Corp, 1915. Film.

The Celluloid Closet. Dir. Rob Epstein and Jeffrey Friedman. TriStar Pictures, 2001. DVD.

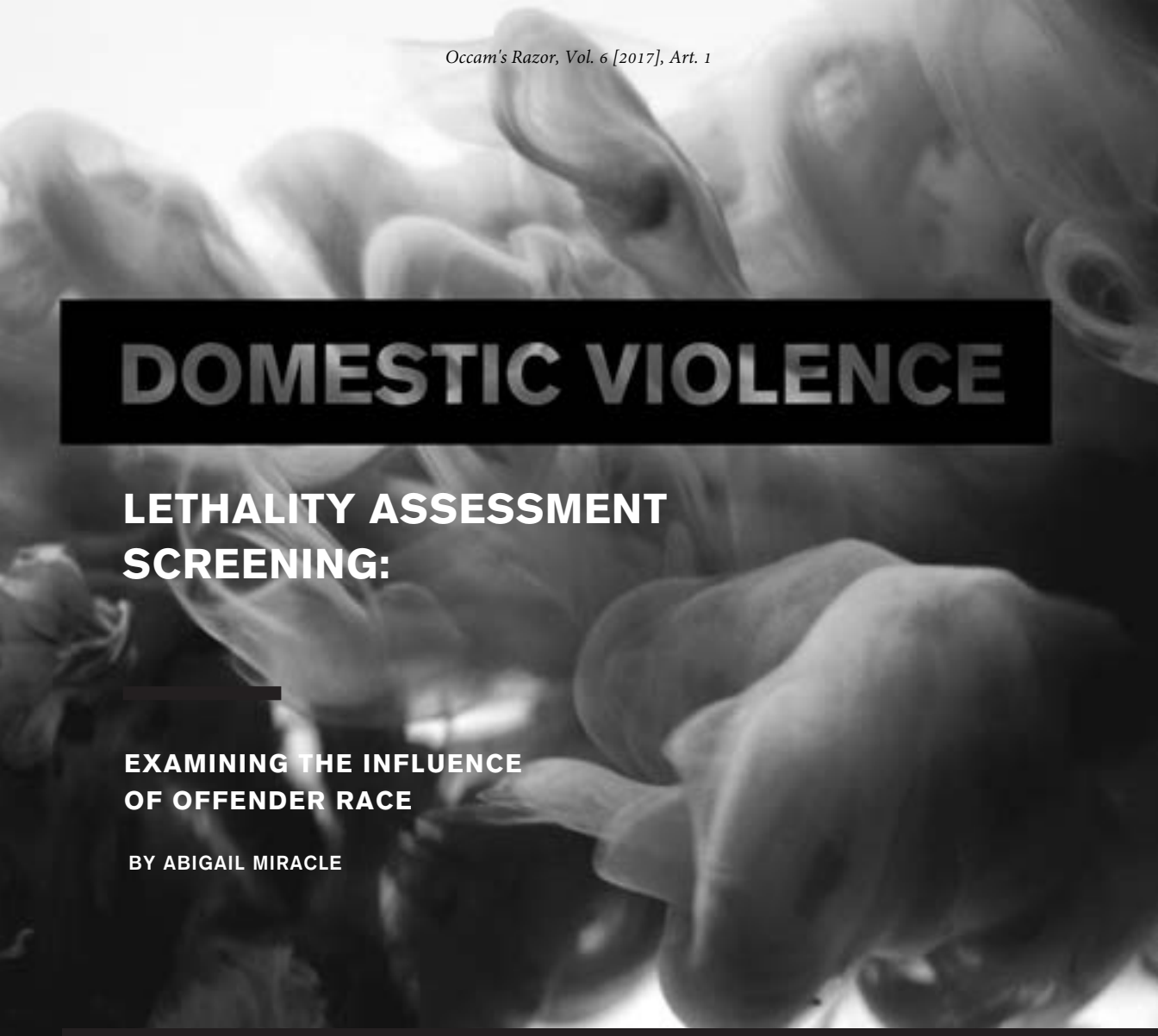
The Mask. Dir. Chuck Russell. Perf. Jim Carrey, Cameron Diaz, and Peter Riegert. New Line Cinema, 1994. Film.

The Silence of the Lambs. Dir. Jonathan Demme. Perf. Jodie Foster, Anthony Hopkins, and Lawrence A. Bonney. Strong Heart/Demme Productions, 1991. Film.

Tropic Thunder. Dir. Ben Stiller. Perf. Ben Stiller, Jack Black, and Robert Downey Jr. DreamWorks SKG, 2008. Film.

Wong, Curtis M. "GLAAD's 2014 Studio Responsibility Index Ranks LGBT Representation In Hollywood." The Huffington Post. TheHuffingtonPost.com, 22 July 2014. Web. 30 May 2015.

Photo Credit: Folkert Gorter



LETHALITY ASSESSMENT SCREENING:

EXAMINING THE INFLUENCE OF OFFENDER RACE

BY ABIGAIL MIRACLE

ABSTRACT

The purpose of this study is to determine whether the prevalent racialized patterns in the criminal justice system are present in domestic violence lethality assessments. On the basis of previous evidence that the criminal justice system practices a racialized pattern that disadvantages people of color, this study tests the hypothesis that non-white domestic violence offenders have a greater risk of being accepted for monitoring by the High Risk Response Team than white offenders. To test the hypothesis, data were collected through the researcher's volunteer position at a non-profit organization, and from relevant law enforcement agencies. Findings indicate that non-white offenders have a greater chance of being accepted for High Risk Team monitoring than white offenders, although the correlation is not statistically significant. When controlling for criminal history, this relationship is strengthened, which leads to the conclusion that the association between non-white races and high-risk monitoring is not due to criminogenic factors.

INTRODUCTION

Recently, a number of cities throughout the United States have implemented methods to assess the dangerousness of domestic violence offenders and determine which cases are most likely to end in homicide. These tools help connect high-risk victims to services that can prevent further danger. Even if the victims of high-risk domestic violence refuse services, High Risk Response Teams may still monitor their offenders. These teams are composed of members from various organizations dealing with domestic violence offenders and victims, including law enforcement, probation, and community-based victim advocates. Although risk assessment is intended to keep victims of domestic violence safe, it can easily slip into the realm of racial profiling, which results in biased decisions about individuals.

The social movement Black Lives Matter has increased the visibility of law enforcement's maltreatment of people of color in the United States; the unfair legal treatment toward people of color manifests in high profile cases of police abuse and neglect as well as in the disproportionate representation of people of color in the prison system. It is apparent that racial bias still exists in contemporary American society. The purpose of this study is to examine the presence of racialized patterns in domestic violence risk assessment.

REVIEW OF THE LITERATURE

America has a long history of problematic race relations, which has prompted thorough research on the topic of racism within the criminal justice system. Researchers question why the proportion of incarcerated people of color—specifically African Americans—is so high relative to their representation in the general American population. Although African Americans made up only 12% of the national population in 2010, they comprised 38% of the U.S. prison population (Guerino, Harrison, & Sabol, 2011, p. 26). Is this disparity a result of racial bias within the criminal justice system? Plethoras

IT IS APPARENT THAT RACIAL BIAS STILL EXISTS IN CONTEMPORARY AMERICAN SOCIETY.

of studies have attempted to answer variations of this question, but results are inconsistent. Steffensmeier, Ulmer, and Kramer (1998) examine how age, race, and gender influence the severity of the sentence imposed by judges. Analyzing Pennsylvania's sentencing outcomes for the years 1989–1992, the researchers concluded that people who are young, black, and male are sentenced more harshly than members of other demographic groups (Steffensmeir, Ulmer, & Kramer, 1998). However, age, race, and gender did not affect sentencing decisions equally; race had a weaker effect on sentencing severity than age and gender (Steffensmeir, Ulmer, & Kramer, 1998).

In *The New Jim Crow*, Alexander provides an in-depth view of racial discrimination in the criminal justice system. Alexander asserts that the current pattern of heavily incarcerating black males under the guise of the War on Drugs strips the rights and opportunities of blacks, resulting in consequences

similar to those of the Jim Crow laws. In a broader sense, as toleration for formal institutionalized racism deteriorates, it is replaced by racial bias disguised in the form of unrestrained discretion for officials.

Blatant bigots are not the only people to hold racial biases, as a person can be racially biased and not be aware of it (Alexander, 2010, p. 107). Psychologists have developed tests that measure implicit racial bias and even people who believe they harbor no bias sometimes score as having high levels. One such study by Blair, Judd, and Chapleau (2004) found that people with more stereotypically black facial characteristics are associated with longer sentences. This pattern held for both blacks and whites. To test racial perceptions, Blair, Judd, and Chapleau (2004) presented undergraduate students with photographs of young black and white male inmates from the Florida Department of Corrections and asked the students to determine the degree to which the inmates' features were stereotypically African American. The researchers coded for the criminal histories of the inmates and analyzed the data. Analysis revealed a positive correlation between Afrocentric features and sentence length (Blair, Judd, & Chapleau 2004). These biases flourish when there is not a stable system of checks to prohibit the use of racial bias in the legal setting (Alexander, 2010).

Steffensmeier, Ulmer, and Kramer (1998) cite schemas associating black men with criminality used by judges as the reason why they give young black men the harshest sentences. Rachlinski, Johnson, Wistrich, and Guthrie (2009) found that judges hold implicit racial biases that can influence their sentencing decisions unless they are made explicitly aware of the convict's race, under which circumstance they are able to compensate for their biases when doling out sentences. These researchers measured racial bias in judges by administering the Implicit Association Test to a sample of judges (Rachlinski et al., 2009). To determine whether this bias influences the judges' sentencing decisions, Rachlinski et al. (2009) gave the judges hypothetical cases to analyze, with race subtly suggested, and with the defendant's race explicitly stated. Judges who exhibited a white preference gave the defendant harsher sentences when primed with black-associated words (Rachlinski et al., 2009). With the defendant's race clearly stated, white judges showed no difference in their sentencing of black and white defendants, suggesting that they corrected for racial bias (Rachlinski et al., 2009). Upon reviewing a multitude of research claiming the existence of racism at all levels of the criminal justice system, Baradaran (2013) concluded that judges generally do not use racial bias in sentencing decision despite their biases against the black community. Additionally, Baradaran (2013) found that at the national level, police only demonstrate racial bias in drug related arrests.

Claims of racial discrimination require proof of racist intent, which results in very few convicted cases in court (Alexander, 2010). Tonry (2010) illustrates how American policies and police practice foster the disproportionate arrest rate and harsh sentencing of blacks. Common racial stereotypes, such as the perception of blacks as dangerous, perpetuate discriminatory ideologies. Many Americans, including African Americans, associate black with danger and white with safety (Tonry, 2010). A preference for whites coupled with a long history of white racial dominance explains why racist policies continue to be supported (Tonry, 2010).

Before conducting the current research, it is important to acknowledge the confounding variables suggested by previous scholarly studies on the topic: likelihood of calling the police, and socioeconomic status. Coker (1995) reports that black women were more likely than white women to report their victimization to the police, and that black men who victimized black women are more likely to be arrested than white men who victimize white women. Coker analyzed data from the National Crime Victimization Survey of the years 1987–1992. Restrictions on data resulted in a sample of 1,535 domestic violence incidents. Thus, according to Coker's research, higher arrest rates among black domestic violence offenders appears to be the result of the greater likelihood of the victim calling the police. Coker did not control for socioeconomic status, a factor that other researchers have found to influence the significance of the relationship between race and prevalence of domestic violence/arrest rate. Previous studies suggest that the socioeconomic disadvantages that racial minorities experience in U.S. society influence their risk for domestic violence perpetration and victimization. Benson, Wooldredge, and Thistlethwaite (2003) posit, "The apparent correlation between race and domestic violence is confounded with the different ecological contexts typically occupied by African Americans and whites" (p. 376). Due to a history of racial segregation in housing and employment, African Americans are disproportionately residents of poor urban communities that lack education and employment opportunities. Analysis of the National Survey of Families and Households and the 1990 census support the authors' argument that neighborhood disadvantage is largely responsible for the correlation between race and domestic violence. Thus, high rates of domestic violence amongst African Americans are likely due to lower socioeconomic status. The authors acknowledge that in reality, there is an ecological divide between African Americans and whites that make it difficult to evaluate how neighborhood environment affects both groups of people (2003). Ideally the present study would control for socioeconomic status, but unfortunately this information was unavailable.

According to Kane (1999), who examined predictive factors of arrest for

THE RELATIONSHIP BETWEEN RACE AND ARREST IS STILL LESS THAN CLEAR

domestic violence offenders, race is not a significant indicator of arrest. However, Kane (2003) states, "The relationship between race and arrest is still less than clear" (p.76). Some communities practice mandatory arrest where police responding to domestic violence incidents are required to arrest the perpetrator. Zorza (1994) asserts that although mandatory arrest laws are an effective means of preventing recidivism overall, they are harmful to both black victims and black offenders. Under mandatory arrest laws, black male offenders are sentenced more harshly and are more likely to lose employment or have difficulty finding employment (Zorza, 1994). Black victims affected by mandatory arrest laws are likely to have difficulty finding employment and housing, and thus are more likely to return to their abusers (Zorza, 1994). Additionally, black female victims are more likely to be ostracized by their community under mandatory arrest laws (Zorza 1994). The greater reliance on the criminal justice system exhibited by black victims relative to white victims is met with further negative treatment by the system.

Previous studies have not examined whether racial bias influences the likelihood that domestic violence offenders are designated as high lethality risk. The county that is examined for the current study has been using a research tool to screen for lethality of domestic violence offenders (Sargent, 2011), formally known as the Lethality Assessment Program (LAP), since 2014. The screening consists of eleven yes-or-no questions administered by a law enforcement official; a certain combination of affirmative answers leads to an automatic high-risk designation. Alternatively, if the officer administering the LAP so chooses, they can screen in the victim based on their own intuition. Victims are offered advocacy services based on their screening results, and those who are determined to be a high lethality risk are referred to the High Risk Response Team. From there, the team gathers additional information about the offender and, if possible, from the victim. The High Risk Response Team then decides whether to accept the offender into the program for monitoring.

Messing et al. (2014) examined the effects of the LAP on women in Oklahoma. In this study, researchers worked with the police who administered the LAP to the consenting victims associated with the screening. They found that victims of domestic violence who had LAP intervention were more likely to establish a code to alert friends and family of their danger and more likely to obtain medical care and protective devices, such as pepper spray or mace (Messing et al., 2014). Victims from the intervention group were also more likely to improve the security of their home, go where their partner could not find them, receive a protection order, or have their offender go to jail. Messing et al. (2014) included race of the victim as a variable in their study to compare demographics of the intervention group and the comparison group, and found no significant racial

differences between groups. The sample consisted of 43% white victims and 29% black victims.

Complementing previous research regarding correlations between domestic violence and race, this present study will attempt to determine whether race influences designation of offenders as "high-risk" by the High Risk Response Team. The team is made up of members from various community organizations that encounter domestic violence offenders and/or victims. The discretion used when selecting offenders to monitor for the High Risk Team is an area where racial bias can come into play; in the case of LAP screening, police are given the opportunity to practice discretion by checking the "screened in by officer's belief" box and designating the offender as high-risk. Previous research suggests that many people, including those that work in the criminal justice system, hold implicit racial biases that associate blackness with danger. This study will be a new contribution to the literature because it is the first study to examine whether race influences the likelihood that domestic violence offenders are designated as high lethality risk.

HYPOTHESES

The first hypothesis states that non-white domestic violence offenders will be more likely to be accepted for monitoring by the High Risk Team than white offenders. The second hypothesis states that the association between offender race and High Risk Team monitoring will not be explained by factors pertaining to criminal history.

DATA AND METHODS

The data used in this study were collected from a county in the Pacific Northwest. The LAP reports were accessed by a researcher and used to obtain information about the offenders designated as high-risk and accepted to be monitored by the High Risk Team. To obtain information about the control group of offenders, the researcher requested a random sample of police reports involving domestic violence calls from the county's two largest law enforcement agencies. Using names and birthdates included on the law enforcement reports, the researcher looked up arrest histories of the offenders electronically. Although the LAPs and reports contain information about both the domestic violence offender and victim, the unit of analysis for this study will be the individual domestic violence offender.

Between November 2014 and March 2016, the High Risk Team designated 24 offenders high-risk. In order to determine whether offender race influences a designation as high-risk, a comparison group of domestic violence cases that were not designated as high-risk was needed. To obtain a control sample, the researcher compiled a complete list of the LAPs administered in the county for January through September of 2015 (n=427). The county's law enforcement jurisdictions implemented the LAP within the past two years. Two local

jurisdictions were associated with the vast majority of the LAPs; the police station for the main city provided 134 LAPs for this portion of 2015, and the county sheriff's office provided 213 LAPs, whereas the highest LAP count for the police stations of peripheral towns was 20 for the same time period. Due to this great divide, only cases from the city police station and the county sheriff's office were considered in analysis. An online random number generator was used to select two sets of 25 numbers: one for the police LAPs and one for the sheriff LAPs. The researcher requested the corresponding reports for these 50 LAPs from the police departments. Due to redacted information and pending reports, only 31 domestic violence offenders were used in the control sample. The present study focuses exclusively on offender characteristics. The selected random sample of 31 cases represents the population of domestic violence offenders who experienced law enforcement contact in the county but were not accepted for monitoring by the High Risk Response Team. The total population of the county is over 200,000.

To construct a dataset for this study, a case number was assigned to each case to keep the offenders' and victims' identities confidential. The main dependent variable in the study indicates whether the offender is monitored by the High Risk Response Team and is entered as a dummy variable where (1 = offender is monitored by team) and (0 = offender is not monitored by team). The main independent variable, offender race, was entered as a dummy variable where (1 = white) and (0 = non-white). Potential race differences in domestic violence offences are controlled for by randomly selecting a control group of domestic violence offenders from the pool of arrested offenders.

For each case, every answer on the LAP was entered by code into the dataset as a dummy variable where (1 = yes) and (0 = no/unknown). Each affirmative answer is indicative of lethality risk. LAP questions representing lethality risk factors are stated as follows:

1. Has he/she ever used a weapon against you/ threatened you with a weapon?

2. Has he/she threatened to kill you or your children?

3. Do you think he/she might try to kill you?

4. Does he/she have a gun or can he/she get one easily?

5. Has he/she tried to choke you?

6. Is he/she violently or constantly jealous or does he/she control most of your daily activities?

7. Have you left him/her or separated after living together or being married?

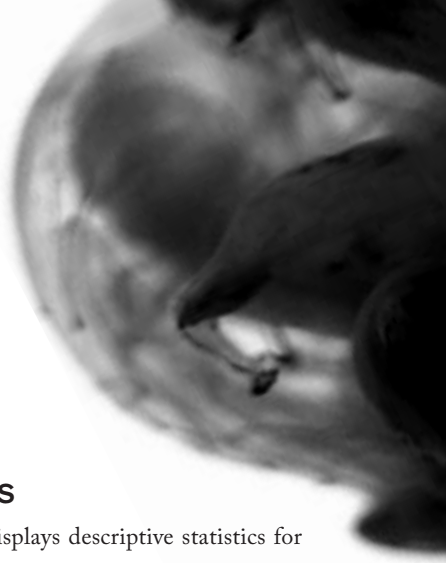
8. Is he/she unemployed?

9. Has he/she ever tried to kill himself/herself?

10. Do you have a child that he/she knows is not his/hers?

11. Does he/she follow or spy on you or leave threatening messages?

Dummy Variable
a variable that can have two possible values: 0 or 1.



If the victim answers "yes" to any of the first three questions or to any four of the last eight questions, the victim screens in due to protocol. These eleven question variables were used to compute a composite variable indicating the offender's LAP score. The LAP score ranges from zero to eleven with zero indicating the lowest level of potential dangerousness and eleven indicating the highest level of potential dangerousness. The two questions that are included on the LAP, but do not influence high-risk designation are "Is there anything else that worries you about your safety?" and "Was he/she using drugs or alcohol at the time of the incident?" Variables for these questions were also coded as dummy variables where (1 = yes) and (0 = no). Another variable, SCREEN, indicates whether the offender screened in as high-risk according to protocol (1), by officer belief (2), or not at all (3). The variable screen was recoded to be a dummy variable where (1 = offender screened in) and (0 = offender did not screen in).

Demographic information from the law enforcement reports for both the offenders and victims was entered into the dataset. Victim race was entered as a dummy variable where (1 = white) and (0 = non-white). Offender gender was coded as a dummy variable where (1 = male) and (0 = female). Victim gender was entered as a dummy variable in the same fashion. Ages of offender and victim in 2015 were entered numerically.

Additional variables include two dummy variables indicating whether the offender was involved in a protection order. One variable is for civil protection orders in which the victim has actively pursued the order (1 = civil protection order present) and (0 = no civil protection order), and the other is for criminal no-contact orders which are instituted by the court without the agreement of the victim (1 = no-contact order present) awnd (0 = no no-contact order). Another variable indicates the number the offender's previous arrests, which ranges from zero to 34 arrests.

RESULTS

Table 1 displays descriptive statistics for the variables measuring dangerousness in this study. Due to the redacted information from the high-risk cases, Table 1 does not represent the full sample. One explanation could be that the greatest proportion of offenders were accepted for monitoring during the first few months that the High Risk Response Team began meeting when the LAP protocol implementation was slow and some law enforcement agencies did not send LAP records to the team. For the 24 cases in which LAP protocols were available, 60% of the victims in the sample answered that their offender is jealous and controlling (n = 23). Forty-six percent of victims answered that their offenders spy on them or leave them threatening messages (n = 17). Fifty percent of the samples screened in as high-risk with the LAP according to protocol (n = 18) and 6 percent of the sample screened in as high-risk with the LAP based on the belief of the officer (n = 2). Only 44 percent of the sample did not screen in as high risk using the LAP. Information regarding arrests and protection orders is included for all offenders in the sample, but since offenders may have arrests and

Variable	Description	Mean	S.D.
LAP VARIABLES	**1 = YES**		
Weapon Use	Whether offender ever used a weapon agaisnt victim or threatened victim with a weapon	.22	.42
Threat to Kill	Whether offender has threatened to kill victim or victim's children	.30	.46
Might Kill	Whether vicitim thinks offender might kill him/her	.14	.35
Gun Acress	Whether offender has access to a gun	.33	.48
Choke	Whether offender has tried to choke victim	.35	.48
Jealous and Controlling	Whether offender is violently or constantly jealous or tries to control most of victim's daily activities	.62	.50
Separated	Whether victim and offender have been separated after living together or being married	.44	.50
Unemployed	Whether offender is unemployed	.41	.50
Suicide	Whether offender has ever tried to commit suicide	.27	.45
Child	Whether victim has child that offender knows is not his/hers	.11	.31
Spy	Whether offender follows or spies on victim or leaves victim threatening messages	.46	.511
Protocol	Whether victim screened in with LAP according to protocol	.50	.51
Officer Belief	Whether victim screened in with LAP according to the belief of the officer	.06	.23
Did Not Screen In	Whether victim did not screen in by LAP or officer belief	.44	.50
Drug & Alcohol	Whether offender was using drugs or alcohol	.50	.51
Arrests	Previous number of arrests for offender	5.11	7.22
Criminal No Contact Order	Whether offender has current criminial no-contact order	.52	.50
Civil Protection Order	Whether offender has a current civil protection order	.24	.38

TABLE 1. Descriptive statistics for dangerousness variables in the analysis of domestic violence lethality assessment, 2015.

NUMBER OF CASES = 24

protection orders in different states that are not accessible within the state system used for this study, the information may not be complete. Offenders who are recorded as having zero protection orders may have protection orders that were not accessible to the researcher. The average number of previous offender arrests is five. Fifty-two percent of the offenders in this sample have criminal protection orders (n = 27) and 24 percent of the offenders have civil protection orders (n = 12).

Descriptive statistics for the variables measuring demographic characteristics of domestic violence offenders are displayed in Table 2. The information is presented separately

Variable	Description	N	Mean	S.D.
HIGH RISK TEAM MONITORED				
Focal Independent Variable				
Race of Offender	Whether offender is white (1=yes)	24	75	.44
Race of Victim	Whether victim is white (1=yes)	14	.86	.36
Additional Independent Variables				
Age of Offender	Offender's age in years	24	34.63	10.48
Age of Victim	Victim's age in years	13	31.31	7.69
Gender of Offender	Whether offender is male (1=yes)	24	1.00	.00
Gender of Victim	Whether victim is female (1=yes)	20	1.00	.00
CONTROL GROUP				
Focal Independent Variable				
Race of Offender	Whether offender is white (1=yes)	31	.84	.37
Rave of Victim	Whether victim is white (1=yes)	34	.79	.41
Additional Independent Variables				
Age of Offender	Offender's age in years	29	41.07	13.85
Age of Victim	Victim's age in years	17	38.94	12.46
Gender of Offender	Whether offender is male (1=yes)	31	.71	.41
Gender of Victim	Whether victim is female (1=yes)	34	.59	.50

TABLE 2. Descriptive statistics for demographic variables in the analysis of domestic violence lethality assessment, 2015.

for the group monitored by the High Risk Team and for the control group. Of the 24 high-risk monitored offenders, 75% are white and 26% are non-white. Of the 31 control group offenders, 84% are white and 16% are non-white. Of 14 victims of monitored offenders, 86% are white. For the control group 79% of 34 victims are white. The average offender age for the monitored group is 35 years with a standard deviation of 11 years. The average victim age is 31 with a standard deviation of 8 years. Among the control group, the average offender age is 41 years with a standard deviation of 14 years. In the monitored group, 100% of the offenders are male and 100% of the victims are female. There is more gender variation in the control group; 71% of the offenders are male and 59% of the victims are female.

Table 3 displays cross tabulations of the lethality assessment variables by

race of offender. Almost 41 percent of white domestic violence offenders versus 54.4 percent of the non-white offenders are monitored by the High Risk Team. Although this 14 percent difference is not significant, these findings indicate that the High Risk Team is more likely to monitor non-white offenders than white offenders. Offenders can be deemed dangerous via the LAP either by protocol (the victim answers "yes" to a certain number of questions) or by the officer's belief. Forty-eight percent of the white offenders in this study screened in due to protocol and none screened in due to officer's belief. Of non-white offenders, 16.7 percent screened in due to protocol and 33.3 percent screened in due to officer's belief. This finding suggests that officers are more likely to perceive danger when responding to domestic violence calls with non-white than white offenders. However, these results must be viewed with caution because of the small number of cases that screened in on the basis of officer belief (n = 2).

To explore the influence of criminogenic factors on race differences in the designation of domestic violence offenders as high danger, the researcher ran a t-test with the offender's LAP score, number of prior arrests, and race, as well as cross tabulations between protection orders and race. These results are presented in Table 4. Among white offenders, the mean number of prior arrests is 5.82, but for non-white offenders, the mean number of prior arrests is only 3.50. This finding contradicts the literature, which suggests that people of color tend to experience higher rates of arrest than whites, but the difference is not statistically significant. Whites in this study also have a higher LAP score than non-whites with a mean score of 3.89 compared to the mean score of 1.50 for non-white offenders. The difference between LAP scores is statistically significant. These findings suggest that the victims of white offenders are significantly more likely than the victims of non-

TABLE 3. Cross tabulations for variables in Domestic Violence Lethality Assessment Study, 2015.

Lethality Variable	Race Variable			Chi-Square	df
High Risk Team	White	Non-White	Total		
High Risk Team Monitored	40.9%	54.4%	43.6%	0.67	1
Not High Risk Team Monitored	59.1%	45.5%	56.4%		
	100.0%	99.9%	100.0%		
LAP Screening					
Screened in with LAP by Protocol	48.3%	16.7%	42.9%	11.27**	3
Screened in with LAP by Officer Belief	0.0%	33.3%	5.7%		
Did not Screen in with LAP	44.8%	50.0%	45.7%		
	100.0%	100.0%	100.0%		

NUMBER OF CASES = 55

Criminality Variable	White	Non-White	t	N
Number of Prior Arrests	5.82	3.50	.904	54
LAP Score (0-11)	3.89	1.50	2.64*	34
			Chi-Square	
Civil Protection Order	22.7%	27.3%	.101	13
Criminal No-contact Order	52.3%	63.6%	.46	30

*p<.05, **p<.01, ***p<.001.

TABLE 4.
Criminal history by race of offender t-test and cross tabulations

	Model 1		Model 2	
Focal Independent Variable	b	Exp(b)	b	Exp(b)
Offender is White	-0.368	.692	-0.856	0.354
Control Variables				
Number of Arrests			.254*	1.290
Civil Protection Order			.267	0.289
Criminal No-contact Order			3.384***	29.491
Intercept	0.000	1.000	-2.724*	0.066
Nagelkerke R^2	0.007		0.527	

*p<.05, **p<.01, ***p<.001.

NUMBER OF CASES = 54

TABLE 5.
Results of logistic regression analysis predicting the log-Odds of offenders being monitored by the High Risk Response Team: Lethality Assessment Study 2015.

white offenders to report that the perpetrator has engaged in behaviors identified as high danger on the LAP tool.

Civil protection orders require the victim to request the order be issued. Twenty-three percent of the white offenders in the study have known current civil protection orders and 27.3 percent of non-white offenders in the study have known civil protection orders. Criminal no-contact orders are issued by the state and are usually associated with domestic violence charges. Fifty-two percent of white offenders and 64 percent of non-white offenders in the study have current criminal no-contact orders. These differences are not statistically significant, but they do suggest that non-white offenders are more likely to have protection orders in place. If the presence of protection orders influences the designation of offenders as high risk, this difference might explain why non-white offenders are more likely than white offenders to be accepted into the High Risk Response Team monitoring program.

Table 5 displays results of logistic regression. This technique allows for the introduction of control variables in the assessment of the impact of race on the designation of high-risk among domestic violence offenders. Results for Model 1, which includes only the key independent variable of offender race, suggest that white offenders are less likely than non-white offenders to be monitored by the High Risk Team. The odds of being monitored by the High Risk Response team are 30 percent lower among white offenders as compared to non-white offenders (Exp. B = .692, p > .05). In Model 2, criminal history control variables are added to

the analysis. By controlling for known factors of criminal history, the effect of race becomes stronger. The odds of being monitored are 65 percent lower for white offenders compared to non-white offenders after control variables for prior number of arrests and protection orders are included (Exp. B = .354, p > .05). Criminal history plays a larger role than race in determining high-risk team involvement. Criminal no-contact orders and arrests have strong positive, statistically significant effects on team monitoring. The effect of civil protection orders is insignificant. In Model 2, control variables are introduced including number of arrests, whether or not the offender has a civil protection order, and whether the offender has a criminal no-contact order or a civil protection order. The Nagelkerke R2 value indicates that the ability to predict High Risk Monitoring is increased 53 percent with knowledge of race, previous arrests and protection orders.

These results indicate that non-white domestic violence offenders have a greater likelihood of being monitored by the High Risk Team than white offenders. Because of the small number of cases (n = 55), this finding is not significant. Factors indicating offender's criminal history are positively associated with High Risk Team monitoring, but they do not explain the relationship between race and High Risk monitoring.

CONCLUSION

The analysis in this study is consistent with previous research that found patterns of race effects in the criminal justice system. Following the pattern, the High Risk Team disproportionately accepts non-white offenders for monitoring. This pattern suggests that racial bias may be influencing the team's decisions as to whom they accept for monitoring. The relationship between monitored offenders and race could be due to factors other than bias that could not be measured in the current study. This study would benefit from being repeated with a larger sample size. The results that were not statistically significant in this study may be significant if a larger sample size were used for the control group. Due to the complicated nature of domestic violence and racial biases, the whole story cannot be portrayed through quantitative analysis. Qualitative research would enrich the understanding of the patterns found in quantitative studies such as this one.

This study is intended to help recognize systematic racialized patterns that may often go unrecognized since they are embedded in the criminal justice system and our society at large. The value of this study is that it may help to raise awareness of ingrained racialized patterns. Results from this study may be used to inform the High Risk Team and other criminal justice system agents that there is a pattern of disproportionate racial representation in domestic violence offenders who are deemed dangerous by the system. In order to eliminate unfair treatment of non-white individuals, we must first acknowledge present biases and act accordingly to correct patterns that disadvantage people of color.

REFERENCES

Alexander, M. (2010). *The New Jim Crow: Mass Incarceration in the Age of Colorblindness.* New York: The New Press.

Baradaran, S. (2013). Race, Prediction, and Discretion. *The George Washington Law Review,* 81(1), 157–222.

Benson, M.L, Wooldredge, J., Thistlethwaite, A.B., & Fox, G.L. (2004). The Correlation between Race and Domestic Violence is Confounded with Community Context. *Social Problems,* 51(3), 326–342.

Blair, I.V., Judd, C.M., & Chapleau, K.M. (2004). The Influence of Afrocentric Facial Features in Criminal Sentencing. *Psychological Science,* 15(10), 674 –679.

Coker, A.L. (1995). Police Involvement in Domestic Violence: The Interactive Effects of Victim Injury, Offender's History of Violence and Race. *Violence Victims,* 10(2), 91–106.

Guerino, P., Harrison P.M., & Sabol, W.J. (2011). Prisoners in 2010. *U.S. Department of Justice, Bureau of Justice Statistics,* NCJ 236096, 1–38.

Kane, R.J. (1999). Patterns of Arrest in Domestic Violence Encounters: Identifying a Police Decision-Making Model. *Journal of Criminal Justice,* 27(1), 65–79.

Maryland Network Against Domestic Violence (2011). *Lethality Assessment Program Maryland Model for First Responder: Learning to Read the Danger Signs.* Bowie, MD: Sargent, D.M.

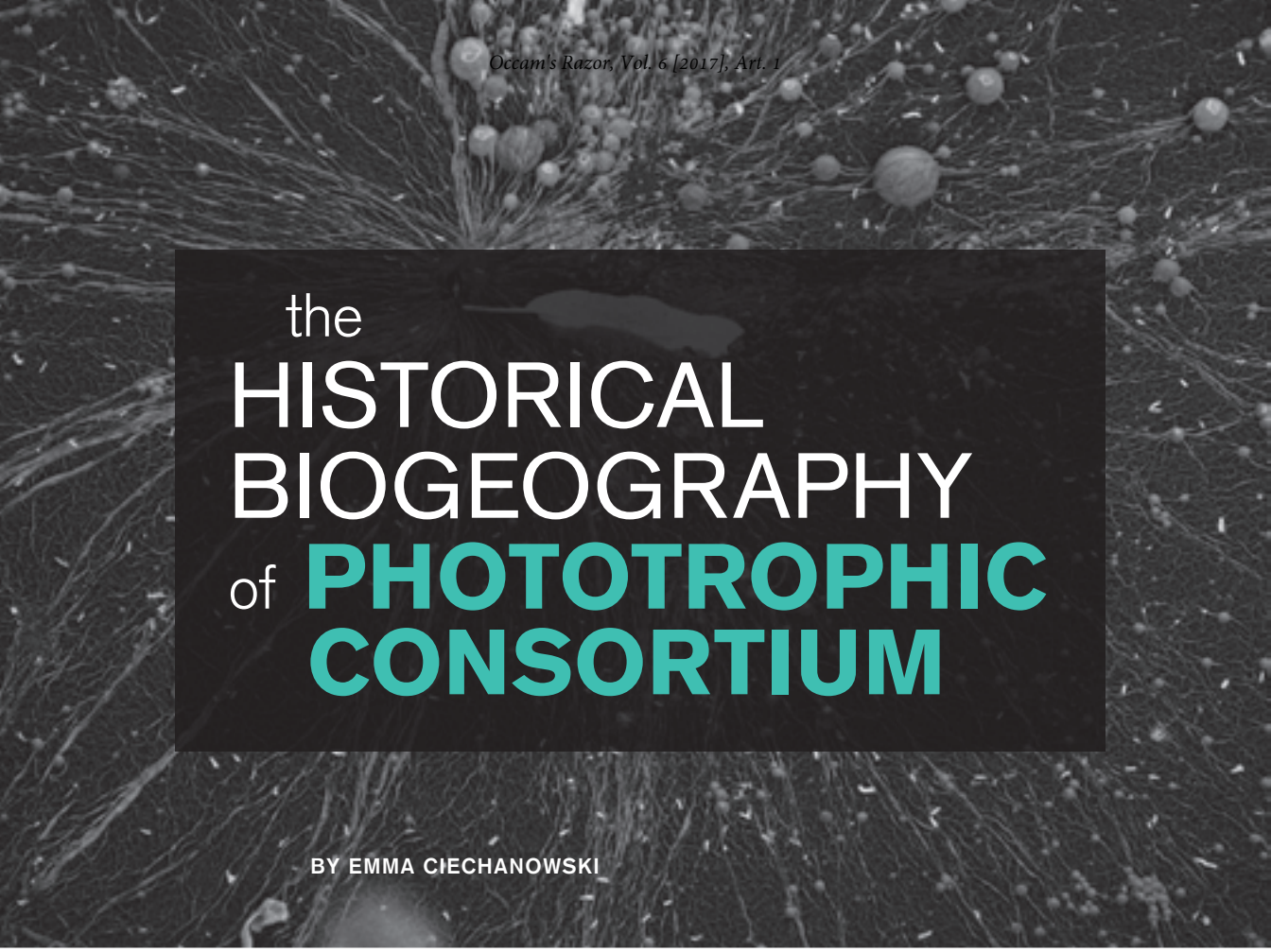
Messing, J.T., Campbell, J., Wilson, J.S., Brown, S., Patchell, B., & Shall, C. (2014). *Police Departments' Use of the Lethality Assessment Program: A Quasi-Experimental Evaluation* (Document No, 247456). Washington, DC: U.S. Department of Justice, National Criminal Justice Reference Service.

Rachlinski, J.J., Johnson, S.L., Wistrich, A.J., & Guthrie, C. (2009). Does Unconscious Racial Bias Affect Trial Judges? *Notre Dame Law Review,* 84(3), 1195–1244.

Steffensmeier, D., Ulmer, J., & Kramer, J. (1998). The Interaction of Race, Gender, and Age in Criminal Sentencing: the Punishment Cost of Being Young, Black, and Male. *Criminology,* 36(4), 763–798.

Tonry, M. (2010). The Social, Psychological, and Political Causes of Racial Disparities in the American Criminal Justice System. *Crime and Justice,* 39(1), 273–312.

Zorza, J. (1994). Must We Stop Arresting Batterers?: Analysis and Policy Implications of New Police Domestic Violence Studies. *New England Law Review,* 28(4), 929–990.



the HISTORICAL BIOGEOGRAPHY of PHOTOTROPHIC CONSORTIUM

BY EMMA CIECHANOWSKI

INTRODUCTION

A consortium is a close physical association between microbial cells of different lineage (Liu et al. 2013). The phototrophic consortium is an aggregation of two different lineages of bacteria: one large motile bacterium surrounded by many smaller photosynthetic bacteria. They live in freshwater lakes around the world with varying degrees of morphological differences. Bacteria are historically accepted as a ubiquitous species, meaning that if the environment permits them to live there, they will. This hypothesis was first introduced by Beijerinck and Becking in the early 1900s to describe the distribution of microorganisms, with the official mantra being, "everything is everywhere, but the environment selects" (Becking 1934, 15). Although this has proven true with common pathogenic bacteria such as *E. coli* and *Salmonella*, studies suggest that conditions do not always permit this hypothesis to hold true.

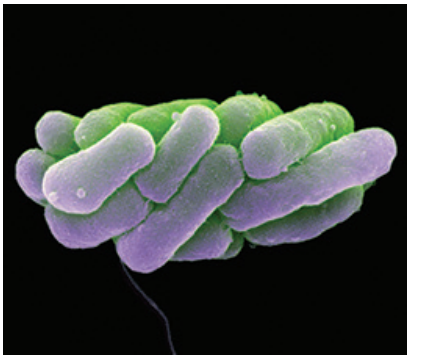
With the similarities in environment that the phototrophic consortia inhabit, and the striking parallels between the evolved interspecific interactions, it would make sense that they are all descendants of one symbiotic partnership, and have diverged morphologically over time within a region. However, the morphological differences that have been found to exist between the geographic distributions of bacteria may indicate separately evolved symbiotic relationships. I believe that the increased fitness—greater ability to survive and divide—provided by the consortium of the two bacterial genera was enough to drive the same symbiotic evolution across separate geographic locations.

SYMBIOTIC INTERACTION

Interspecific interactions have the ability to increase the fitness of one or both organisms. These interactions come in a variety of flavors, with the differences being whether one or both of the organisms are benefiting from the interaction. Interspecific interactions also have varying degrees of proximity, from living completely separate from each other and interacting sparsely, to living inside of one another. When one organism lives in close contact with another, it is called a symbiosis. This close association between organisms allows for a powerful reciprocal selection to occur, resulting in coevolution. This is one of the more beautiful artifacts of evolution because through cooperative symbiosis, the throes of time, and the environment, the resulting organisms—by varying degrees—need each other. For bacteria, this is not an uncommon trait. One example is the rumen bacteria possessed by cows, which live in the digestive tract and help digest the consumed plant matter in exchange for nutrients. Interbacterial mutualisms are common with varying degrees of codependence. Many bacteria are able to thrive in wildly diverse communities, utilizing important metabolites from the metabolic waste of others. In layman's terms, one man's trash is another man's treasure. The phototrophic consortium that I will address here is exemplary of a mutually beneficial symbiotic relationship.

ECOLOGY, MORPHOLOGY AND PHYSIOLOGY

Since their discovery over one hundred years ago, there have been seven described morphologies of the phototrophic consortium; all are made up of a single, large, heterotrophic motile bacterium, covered by 13–69 non-motile, phototrophic sulfur bacteria, also called epibionts (Madigan 2015). The central bacterium is motile due to the presence of a monotricious flagellum, though some also have gas vesicles, which allow it to move vertically in the watercolumn (Table 1). The epibionts are either phototrophic green or brown sulfur bacteria, or in one reported case, a combination of both (Table 1).



PHOTOTROPHIC CONSORTIUM

Phototrophic consortia are found worldwide in freshwater lakes and ponds where light reaches areas of water containing hydrogen sulfide (Madigan 2015). In these lakes, the consortia are constantly repositioning in order to maintain adequate light levels for photosynthesis and sulfur metabolism.

Movement of the motile central bacterium directly benefits the non-motile epibionts. The phototrophic consortium differs from motile phototrophic bacteria in that they move away from the light as opposed to towards it, in an exhibition of *scotophobotaxis* (Hays et al. 2015). The phototropic consortium also exhibits chemotaxis towards sulfide and sulfide-containing compounds (Liu et al. 2013). This movement is significant in understanding the symbiosis because the bacteria doing the movement is neither phototrophic, nor is it a sulfur bacteria. Movement done by the central bacteria is to serve the needs of its phototrophic sulfur-loving passengers. The mutual benefit of the symbiosis between the two is a ride in exchange for some food.

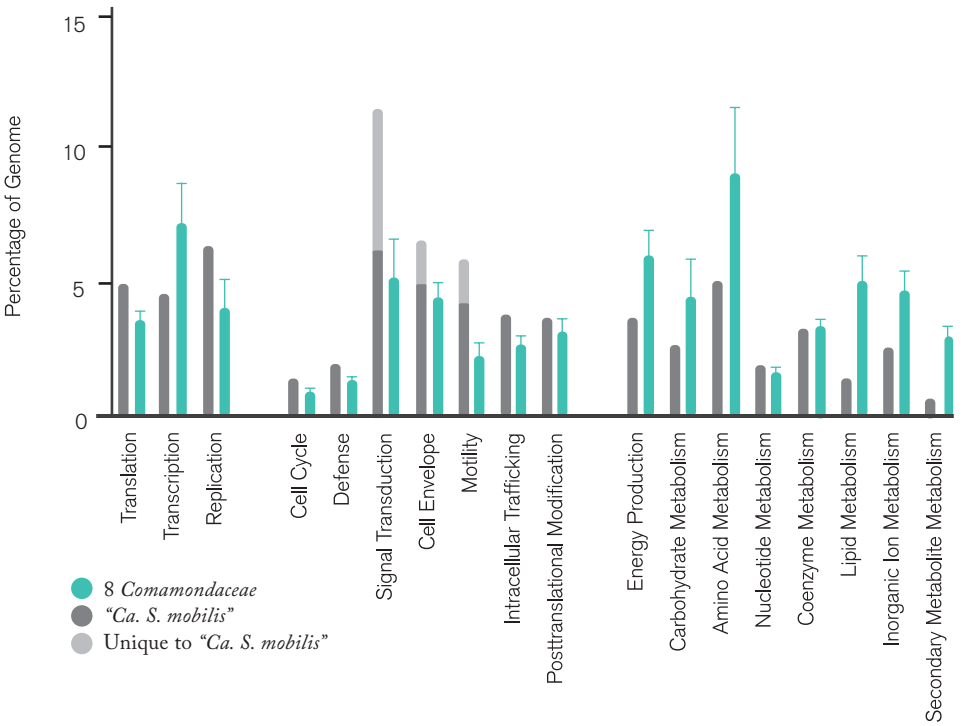


FIGURE 1.
A comparison of the gene contents of the central bacterium of the phototrophic consortium *"Ca. S. mobilis"* and eight of its non symbiotic relatives. Green bars indicate the average percentage of the genome belonging to the eight relatives, gray bars indicate the percentage of the genome belonging to *"Ca.S. mobilis"*, and light gray bars indicate the percentage of the genome that is unique to the *"Ca.S. mobilis"* (Liu et al, 2013).

In order to understand the biogeographic relationship between the different populations of the phototrophic consortium and their evolution, it is important to understand the depth of their symbiosis; the phototrophic consortium represents one of most interdependent relationships between two unrelated bacteria. They have melded many of their physiological processes, are metabolically coupled, and have coordinated their cell division. Even the names of the organisms, which are purely taxonomically based, are put in quotation marks due to the fact that they are a mixed culture and not a true species (Hays et al. 2015). In fact, the central bacterium is likely no longer capable of independent growth; so far, it has been impossible to independently culture without the help and essential metabolites from its phototrophic partner. This is due to a massive gene loss during its symbiotic evolution, especially for genes involved in metabolism (Liu et al. 2013). An example of this gene loss is in the *"Candidatus Symbiobacter mobilis"* (*"Ca. S. mobilis"*), the central bacterium from the most studied consortium, *"Chlorochromatium aggregatum."* The lost genes were identified by comparing eight different non-symbiotic genomes from the *Comamonadaceae* family with that of the *"Ca. S. mobilis."*

In addition to losing genes, the *"Ca. S. mobilis"* also gained some that were not found in the other eight *Commanonadaceae* genomes (Liu et al. 2013). These genes were mainly involved in signal transduction, cell envelope biogenesis, and cell motility (Figure 1). This is likely due to the increased need for intercellular communication and motility, and is indicative of a long-term coevolutionary relationship. Due to the high specificity between the bacteria, it would seem more probable that the symbiosis evolved once, rather than multiple times, supporting the hypothesis of a symbiotic ancestor followed by distribution. However, short generation times increases the speed of evolution in bacteria. If the symbiosis is highly beneficial and dramatically increases fitness, it is possible that the phototrophic consortium evolved separately, multiple times.

SLOW CLOCK ANALYSIS OF rRNA

Depending on the protein or molecular marker being examined, phylogenies can give differing relative estimates of time. Based on the protein being examined, one can look at interrelatedness based on "fast clock" or "slow clock" analysis. The less essential a gene is, the more likely it is to be a fast clock gene. Its ability to accumulate mutations and evolve is increased because it is less constrained by purifying selection. Slow clock genes are more ancient and essential; they withstand mutations due to higher levels of purifying selection and thus have higher levels of conservation.

Ribosomal RNA (rRNA) is often used to analyze interspecific relatedness for many reasons. First, it exists in all living organisms, which indicates that it is highly conserved; this makes the variable regions much more significant. Second, it is not involved in horizontal gene transfer, which is important because we can definitively say that the phylogenetic relationships are due to ancestry— or lack thereof—and not part of a randomly acquired gene. Third, there is already a rather large database, so the comparison and sequence alignment of rRNA with other species and organisms is easy to perform, and can give a wider picture of relatedness without having to experimentally examine an entire organism's family.

ANALYSIS OF EPIBIONT rRNA

The phylogenetic relationships between the different "species" in different lakes of epibionts were investigated by analyzing the 16S rRNA. This was completed in a 2000 study by Fröstl and Overmann. The study investigated the relationship between the epibionts of *"C. aggregatum"* from Lake Dagow in Germany and the epibionts of *"C. aggregatum," "C. glebulum,"* and *"C. magnum"* from Lake Echo in Washington, U.S.A. So far only the epibionts have been able to be cultured, due to the high sensitivity and dependence of the central bacterium (Stewart 2012). Comparative analysis of the 16S rRNA was done through PCR amplification and sequencing, followed by sequence alignment. The 16S rRNA gene fragments of the different sulfur bacteria

SHORT GENERATION TIMES INCREASES THE SPEED OF EVOLUTION IN BACTERIA

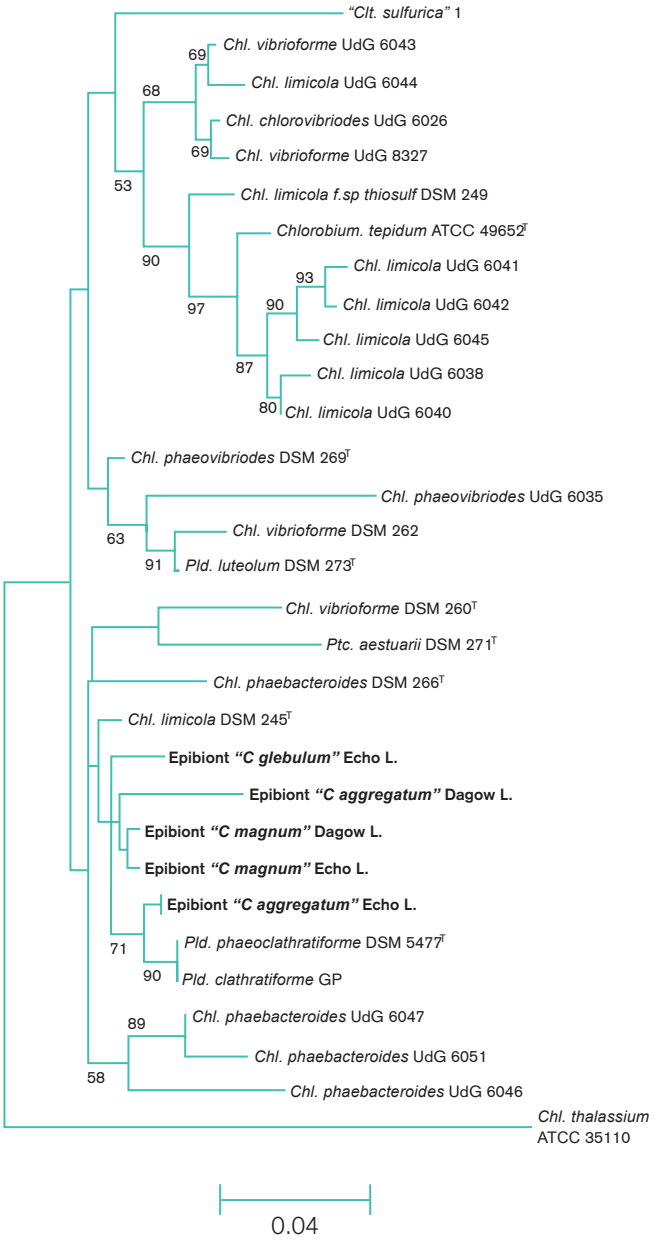


FIGURE 2.
A phylogenetic tree of the five different epibionts from Lake Dagow and Lake Echo, along with known radiations of non-symbiotic green sulfur bacteria. The bar at the bottom indicates 0.04 fixed point mutations per base (Fröstl and Overmann, 2000).

were analyzed by denaturing gradient gel electrophoresis, which yielded single, unambiguous bands (Fröstl and Overmann 2000).

Fröstl and Overmann's study found that the banding analysis of the denaturing gradient gel in the 16S rRNA sequence found that the epibionts from *"C. aggregatum,"* *"C. glebulum,"* and *"C. magnum"* from Echo Lake had high similarity compared to the epibionts *"C. aggregatum"* and *"C. magnum"* from Dagow Lake (Fröstl and Overmann 2000). This showed that cells of a specific morphotype of consortia living in a single lake belong to the same phylotype.

Phylogenetic analysis of the 16S rRNA found that the epibionts form a cluster in the sulfur bacterial radiation with six other free-living strains (Fröstl and Overmann 2000). This analysis supports the idea that although they are closely related, there is no singular ancestral appearance of the symbiotic trait (Figure 2). Additionally, the phylogenetic analysis of the epibionts from the consortia of Lake Dagow and Lake Echo showed that similarity in location was not tied to higher levels in relatedness (Figure 2). This supports the hypothesis that the symbiosis of the phototrophic consortium evolved multiple times in multiple locations.

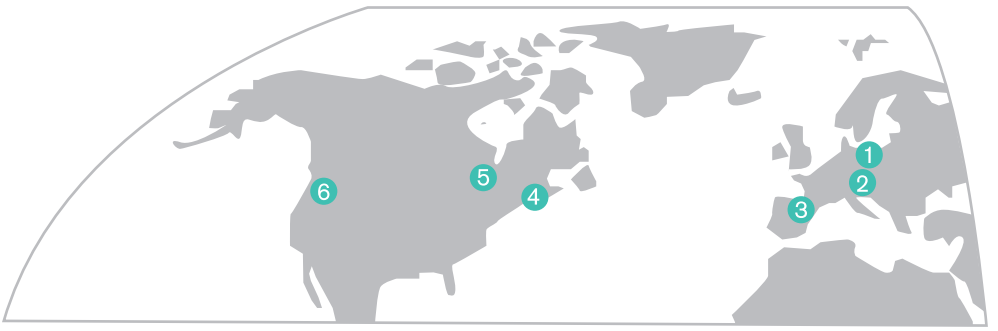


FIGURE 3.
This is a map of the geographic regions where samples of phototrophic consortia were taken. Region 1 and 2 sampled 3 different lakes in Germany. Region 3 sampled two different lakes in Spain. Region 4 sampled one pond in Massachusetts. Region 5 sampled seven lakes in Michigan. Region 6 sampled one lake in Washington (Glaeser and Overmann, 2004).

Another study by Glaeser and Overmann in 2004 examined the phylogenetic relatedness between epibionts of phototrophic consortia from 14 lakes with similar environmental parameters in six different geographical regions: Germany, Spain, Massachusetts, Michigan, and Washington (Figure 3). Like Fröstl and Overmann's study in 2000, this study also used the 16S rRNA as a genetic marker in order to determine the phylogenetic relatedness between the different morphotypes and strains of the epibionts. Out of the seven morphological forms of phototrophic consortia found in the 14 lakes, 15 distinct 16S rRNA sequence types were detected (Glaeser and Overmann 2004). To complicate matters, these distinct 16S rRNA sequence did not always match with specific morphological forms (Table 2). This resulted in a total of 19 different "phylomorphotypes," a term coined to describe the different combinations of morphology and phenotypes (Table 2).

It was found that in a single lake, the matches between phylotype and morphotype would be consistent, whereas on a global scale they were not. The distinct phylogenetic clusters of the phototrophic consortium suggest that the symbiosis either arose independently, or that a common ancestor of the sulfur bacteria was symbiotic. If the latter were the case, then the free-living trait would be the one to have evolved independently, which is unlikely due to the widespread nature of the green sulfur bacteria radiation. The phylogenetic analysis of the 16S rRNA over the six geographic regions showed that there is a nonrandom pattern of

THEREFORE, THE PHOTOTROPHIC CONSORTIUM HAS NOT DESCENDED FROM A SINGLE ANCESTOR.

distribution, with increased differences across continents (Glaeser and Overmann 2004). Therefore, the phototrophic consortium has not descended from a single ancestor.

DISCUSSION AND FINAL THOUGHTS

It has been assumed that microorganisms do not have geographical barriers for distributions, and that given high levels of ubiquity in microorganisms, and a suitable environment, a microorganism will live there. The study of the biogeographic distribution of phototrophic consortia shows us the flaws in these assumptions. There is a nonrandom distribution and little similarity in the epibiont of phototrophic consortia across continents, and higher similarity in populations in neighboring lakes. This provides evidence for a slow dispersal of the phototrophic consortia over large distances. However, due to the nature of microorganisms, the speed and ingenuity of their evolutions make it possible for complicated traits to arise independently in populations. The close association of consortia expands the functional and metabolic abilities of the organisms and allows for a greater ability to survive perturbation, thus increasing the fitness of both organisms (Hays et al. 2015). This increased fitness means that there is a greater likelihood of symbiotic traits becoming fixed in a population. Considering both of these factors and the phylogenetic data from recent studies, I believe that the phototrophic consortium evolved separately across large spatial distances, but experienced dispersal across short spatial distances.

Research on the origins and basis of symbiotic interactions between bacteria is important for an understanding of the wide range of mutualism that exists in microbial communities. The consequence of mutualisms between bacteria is not only beneficial for the bacteria themselves, but also for the stability of ecosystem function. Nestled at the bottom of the food chain, microorganisms are the backbone to any ecosystem. Just as genetic diversity within a population improves that species' adaptability and chance of survival, diversity within the microbial community of an ecosystem helps serve as a buffer for environmental perturbations. Phototrophic consortia are not only of ecological significance in lake ecosystems, but are also excellent model systems for the evolution of the bacterial interactions. Studying their intimate and complex symbiotic relationship helps give us a better understanding of the molecular mechanisms and evolutionary factors that may have led to multicellularity, and hence the diversity of eukaryotic life we see on Earth today.

REFERENCES

Becking B., Lourens G.M. 1934. *Geobiologie of inleiding tode milieukunde.* The Hague, the Netherlands: WP Van Stockum and Zoon.

Cerquada-Garcia D., Martinez-Castilla L.P., Falcon L.I., Delaye L. 2014. Metabolic analysis of *Chlorobium chlorochromatii* CaD3 reveals clues of the symbiosis in *"Chlorochromatium aggregatum"*. *ISME Journal* (8):991-998.

De Wit R., Bouvier T. 2006. 'Everything is everywhere but, the environment selects'; what did Baas Becking and Beijerinck really say? *Enviromental Microbiology* (84):755-8.

Fontaneto D. 2011. Biogeography of Microscopic Organisms: *Is Everything Small Everywhere?* New York: Cambridge UP.

Fröstl J., Overmann J. 1998. Physiology and tactic response of the phototrophic consortium *"Chlorochromatium aggregatum"*. *Arch Microbiol* (169):129-135.

Fröstl J., Overmann J. 2000. Phylogenetic Affiliation of the Bacteria That Constitute Phototrophic Consortia. *Archives of Microbiology* (174):150-58.

Hays S.G., et al. 2015. Better together engineering and application of microbial symbiosis. *Current Opinion in Biotechnology* (36):40-49.

Glaeser J., Overmann, J. 2004. Biogeography, Evolution, and Diversity of Epibionts in Phototrophic Consortia. *Applied and Environmental Microbiology* 70(8):821-830.

Kanzler B.E.M., Pfannes K.R., Vogl K., Overmann J. 2005. Molecular Characterization of the Nonphotosynthetic Partner Bacterium in the Consortium *"Chlorochromatium Aggregatum"*. *Applied and Environmental Microbiology* 71(11):434-441.

Liu Z., et al. 2013. Genomic analysis reveals key aspects of prokaryotic symbiosis in the phototrophic consortium *"Chlorochromatium aggregatum"*. *Genome Biology* (14):R127.

Madigan M.T. 2015. *Brock Biology of Microorganisms*. Boston; Munich: Pearson.

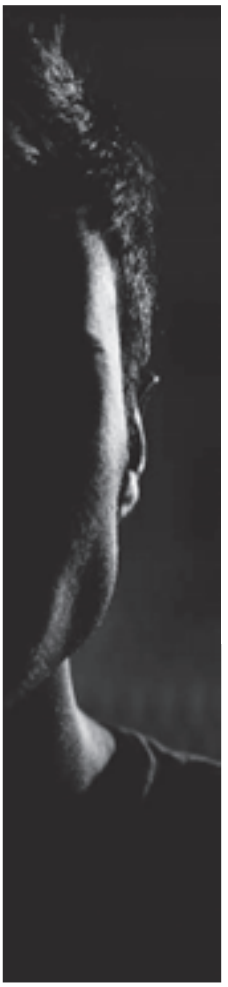
Martiny J.B., et al. 2006. Microbial biogeography: putting microorganisms on the map. *National Review of Microbiology*, (42):102-12.

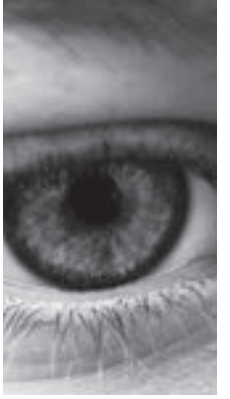
Overmann J. 2010. The phototrophic consortium *"Chlorochromatium aggregatum"*—a model for bacterial heterologous multicellularity. *Advanced Experimental Medical Biology*, (675):15-29.

Overmann J., Van Gemerden H. 2000. Microbial Interactions Involving Sulfur Bacteria: Implications for the Ecology and Evolution of Bacterial Communities. *FEMS Microbiology Reviews FEMS Microbiol Rev* 24(5), 591-99.

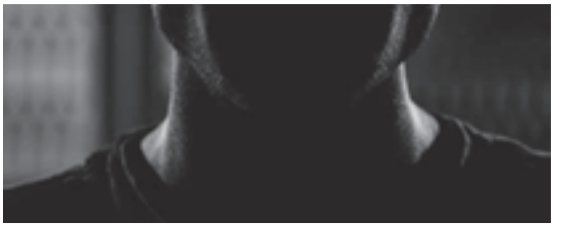
Stewart E.J. 2012. Growing unculturable bacteria. *J Bacteriol*, (194):4151-4160

Ramette A., Tiedje J.M. 2007. Biogeography: an emerging cornerstone for understanding prokaryotic diversity, ecology and evolution. Microbial Ecology, (532):197-207





PERSPECTIVALISM AND BLAMING



BY DANA ANN

ABSTRACT

Using the combination of two views of blame from T. M. Scanlon and J. J. C. Smart, I will support my thesis *perspectivalism*, that blame from the perspective of a third party is fundamentally different than blame from the perspective of an injured party. By presenting examples that illustrate common beliefs concerning hypocrites and cases involving moral luck, I will give reasons as to why perspectivalism has strong explanatory value, and also provide motivation for considering blame as a complex topic requiring a pluralist theory. In doing this, I will show that two statements about hypocrites are true if we accept perspectivalism. First, as many philosophers have noted, hypocrites lose their standing to blame from a third party perspective. Second, with my new understanding of blaming as the injured party, I will conclude that hypocrites retain their standing to blame in the injured perspective in virtue of their relationship to the wrong doing. In the case of the moral luck examples, I will illustrate the complexity that comes from having two types of blame. Ultimately, I will argue that a correct general theory of blame must consider the position of the blamer relative to an instance of wrongdoing, using the explanatory value of the hypocrite cases, without taking a stand on a specific theory of blame.

INTRODUCTION

Imagine a grocery store owner watching in surprise and shock as a thief steals a can of soup and exits the store. It seems right to say that the owner has both the epistemic justification for their belief that they have been wronged, and also the moral standing to blame the thief for that wrong.

Now imagine a second scenario, in which an individual is walking down the street and is carelessly knocked to the ground. To that individual's surprise, their bag has gone missing, along with the person who knocked them down. Again, it seems right to say that the individual on the street has both the epistemic justification for their belief that they have been wronged, and the moral standing to blame the thief for wronging them.

But what if I told you that the thief from the first scenario is the victim in the second? Assuming that they had no permissible excuse for their actions in the initial instance, would it not seem absurd for them to feel anger about being a victim of theft when they were, just the other day, stealing from the grocery store? By becoming upset when their bag was stolen, the thief in the original scenario fits the description of a "garden-variety hypocrite," an individual who "unrepentantly engages in the very activity they're blaming others for" (Coates 2016, 19). We tend to respond to these types of blamers with the comments like "Look who's talking," because of the perceived inconsistency that exists between their actions and expressed blame.

This claim against the hypocrite's standing to blame is a common view shared by many philosophers; for more examples, see Dworkin 2000, Cohen 2012, Wallace 2011.

Perspectivalism
That blame from the perspective of a third party is fundamentally different than blame from the perspective of an injured party.

In spite of the intuitiveness of this response, in this paper I will argue that the common views on hypocrisy correctly interpret the moral standing of the hypocrite as a third party blamer, but fail to recognize the key difference between blaming from a third party perspective and blaming from the perspective of an injured party. I will argue that there is a morally relevant feature of being a member of an injured party that allows the standing of even a hypocritical victim to remain intact; that where an individual is placed relative to an instance of wrongdoing makes a difference to whether their own faults are relevant to their standing to blame. I shall refer to this view as *perspectivalism*.

1. OUR TWO TYPES OF BLAME

The topic of this paper hinges on the claim that blame from the perspective of a person who is not directly—or is indirectly—harmed in the given circumstances, or *third party blamer*, differs crucially from blame that comes from the perspective of a person who has been directly wronged, known as *injured party blamer*. The motivation for this

thesis came from conflicting intuitions that T.M Scanlon and J. J. C. Smart both posit correct theories of blame. For the purpose of my conclusion, I will illustrate how they can be combined to support the theory of perspectivalism. I do not argue in favor of either of Smart's or Scanlon's views; for the sake of this paper I will assume they are correct, as my goal is to show that, for the case of the hypocrite, perspectivalism has both consistency and explanatory power, and shows complexity in the cases involving moral luck.

First, Smart's cognitive theory of blame boils down to placing, not necessarily emotionally, an evaluation that the performer of some action has done something morally wrong in performing said action, and implies they're responsible for the action. This is not to say that emotions do not accompany third party blame, but rather that this type of blame is sufficient in and of itself, without considering emotional aspects. Similar to how an art piece is graded as being good or bad, to blame someone is to place a negative evaluation on that person's action. But unlike the grading of art, the latter type of evaluation implies the individual has a moral responsibility for their action.

For Scanlon,

> ...to claim that a person is blameworthy for an action is to claim that that action shows something about the agent's attitudes toward others that impairs the relations that others can have with him or her. To blame a person is to judge him or her to be blameworthy and to take your relationship with him or her to be modified in a way that this judgment of impaired relations holds to be appropriate. (Scanlon 2008, 125)

Smart argues that blame is a dispassionate and clearheaded response to action, but he acknowledges that most people do not praise or blame in this dispassionate way. Utilizing Scanlon's view of blame, we can find motivations as to why it's not usual to blame dispassionately. As Scanlon suggests, relationships are "constituted by certain attitudes and dispositions" among which "intentions and expectations about how the parties will act toward one another" are most important (Scanlon 2008, 131). To impair the relationship is to damage the expectations of how each party will interact with each other. To blame, then, is to register that damage has been done to the relationship and the subsequent need for modification.

Now, here we see that Scanlon's objection to Smart's understanding of blame is that the latter failed to recognize the sense of force behind blame, or the specific damage done to the injured party, which comes from the issue of treating all blame as being identical. However, if we take perspectivalism to be true, there is no need to require that third party blaming have the same sense of force as does injured party blaming. This means we could grant that third party blaming is, at a minimum, a dispassionate evaluation of wrongdoing that

implies responsibility, while acknowledging that direct harm done to an injured party allows for reactionary blame, which takes into account Scanlon's concept of the force of blame.

Going forward, I will rely on Smart's theory to represent third party blame, and Scanlon's view to represent injured party blame, which will demonstrate the consistency and explanatory power that perspectivalism has in the case of the hypocrite.

2. HYPOCRITICAL BLAME

Imagine the thief who stole the can of soup is later arrested for stealing jewels from a jewelry store, and is sent to prison. Once there, the thief finds that a few personal items are missing. After investigating, the thief comes to the epistemically sound and justified conclusion that the guards have been stealing the items. Enraged by this conclusion, the thief blames the guards for committing this wrong.

The value of this example lies in its ability to illustrate how the victim may appropriately blame the guards, in spite of having unrepentantly engaged in precisely the same sorts of behavior they now blame the guards for. And even though the thief would lose the standing to blame the guards for stealing from other inmates, since that thief has committed a similar act before and is likely to reoffend, they still have the standing to react specifically to the wrongs of which they are the injured party.

Scanlon's view would acknowledge that guards owe inmates specific types of interactions, and that these obligations are grounded in the expectations that guards and inmates may reasonably have of each other, in virtue of the nature of their relationship in the moral community. By stealing from this individual, the guards have modified the relationship between the two parties in a negative way and have thereby impaired the original relationship. The inmate's response of blaming is not simply an acknowledgement of the guards' wrongdoing, but also a reactionary response to the guards' failure to fulfill the expectation of the relationship. This would account for why, had the guards returned the missing items, the inmates would most likely not respond with abandoning their blame.

Now, imagine this same inmate is not the injured party, but rather a third party blamer. It seems that in this circumstance, the guards could respond to their blame with the comment "Look who's talking." Here, such a response is sufficient to highlight the inmate's loss of the moral standing to blame. Since the inmate has no other areas they can criticize, and given that they have not personally suffered harm to any relationship, the inmate's blame now seems to be inappropriate.

Of course, there are circumstances wherein the injured party could involve more individuals than just the direct victim. Another inmate, who is emotionally close to the party by virtue of their intimate relationship, has cause to be a part of the injured party. Moreover, when the warden finds out about the actions of the prison guards, their blame could be considered as that of an injured party, given the expectations regarding the relationship between the warden and the guards.

In comparison, everyone watching the news of the prison guards being caught and arrested for stealing from the inmate, would be blaming from a

third party perspective. Since they are not family members of the guards or of the inmate, their blame would simply be acknowledging the fault in the guard's actions, and implying that the guards are responsible for those wrongdoings. It seems that perspectivalism, using the respective theories of Scanlon and Smart, supports the existence of these two distinct functions of blame.

3. ADDITIONAL COMPLEXITIES CONCERNING MORAL LUCK

Imagine now that, on one unfortunate night, a morally conscious person is driving down a residential street. This individual is a good driver, who takes into account the safety of others and does their best to follow all driving restrictions. On this occasion, even as the driver is paying close attention to their surroundings, a small child runs out into the street, and the driver is unable to stop in time to avoid a collision. Society and the moral community will write this off as an unavoidable accident and hold no one at fault; but what about the child's parents? Can these individuals still blame the driver for the loss of their child, when the moral community finds the driver to be unblameworthy?

My thesis of blame, which combines specifically the views of Scanlon and Smart, would allow for the parents of the deceased child to—in fact—appropriately blame the driver under these exact circumstances. It is true that the driver is not responsible for the child running into the street, nor for the subsequent harm to that child, but the parents are still left with a loss. Even though the driver did not mean for this horrible event to happen, that individual is still the direct cause of the child's death. This is a strong example for many reasons. First, it is honest about the ways in which our society operates, and it has explanatory power; sadly, there are parents who have experienced such tragic events, and there are also people that are involved in such tragic accidents despite being safe drivers. Second, I have the internal motivation to protect the driver from the blame of the parents because I could easily find myself in a similar position. On the other hand, I could just as easily be in the position of the parents, who have lost a loved one. Because both motivations are present, this serves as an intuitive example for the distinction between the blame from the perspective of an injured party versus that of a third party, and gives more reason to believe the two kinds of blame are fundamentally different. Speaking from the third party perspective and as a member of the moral community, I would say that the driver is not responsible for the death of the child because the event was out of their control, and there are no actions of the driver to be criticized; the tragic outcome is merely the result of moral luck. However, if I was speaking from the perspective of the injured party, I would need to acknowledge the loss, and the resulting modification to the relationship of the members. It

Moral Community
In this paper, "moral community" is used to reference how a hypothetical community, bound by an identical moral theory of right action, would evaluate specific events. It is not required, within this essay, to confirm the exact theory of right action, as it relies on basic responses given in the domain of moral responsibility.

Unblameworthy
An evaluation that a moral individual is not responsible or fitting of blame for a given event or act. This paper pushes against the claim that an unblameworthy agent by the moral community is identically unblameworthy by the injured party.

Moral Luck
Area within ethics, which studies the influence of factors that are out of an agent's control in relation to whether that agent can be an object of moral judgement specific to a variety of different categories (actions, character disposition, casual events, circumstantial events, etc.).

is a reactionary expression from the perspective of the parents, but this example as whole, shows the complexity of both perspectives. Although the consequences that result from cases that are influenced by moral luck are controversial, and so my point can be similarly seen as controversial, it is worthwhile to consider the complexity of two different perspectives of blame.

4. CONCLUSION

It seems that our intuitions about blame support the conclusion that some people have greater claims to blaming than others; perhaps this a result of injured party blamers blaming in this very distinct way, specifically in cases concerning where the hypocrite has the standing to blame as the injured party. Smart and Scanlon both offer insight into distinct modes of blame. Of course, other theories of blame may not fit so perfectly with the theory of perspectivalism as those offered by Smart and Scanlon, being that one is a cognitive theory of blame and the other conative. For example, it is not clear to me that emotional theories of blame could accurately fit into this framework. Putting this concern aside, my hope is that, even if one rejects Scanlon's and/or Smart's view, the reader will still be left with motivation to see that there are different types of blame. It is intuitive that victims of events have a different experience than bystanders, whose perspective is that of the third party. Should it not be the case that their reactionary blame would also be different? Ultimately, the ways in which I've examined both the hypocrite cases and the cases involving moral luck provide motivations to question how philosophy of blame has somehow avoided studying pluralistic theories thus far.

REFERENCES

Coates, D. J. "Moral Fragility and the Standing to Blame." Essay, University of Houston, 2016. Unpublished.

Scanlon, T. M., *"Moral Dimensions: Permissibility, Meaning, Blame,"* Cambridge, MA: Harvard University Press. 2008.

"The Significance of Choice." In *The Tanner Lectures on Human Values VIII, Vol. 8*, 149-216. Salt Lake City: University of Utah Press, 1988.

Smart, J. J. C., "Free Will, Praise and Blame." *Mind* 70, no. 279 (1961): 291–306.

NONALCOHOLIC FATTY LIVER DISEASE: CAUSE TO TREATMENT

BY TAVLEEN AULAKH

INTRODUCTION

Imagine two individuals, both suffering from severe liver damage. With excess fat molecules concentrated in the hepatic cells, their livers are inflamed and scarred. These deteriorating livers are also supplementing the development of chronic obesity, diabetes, cardiovascular diseases, and hyperlipidemia. While one of these individuals is a middle-aged male with a long history of alcohol addiction and abuse, the other is only thirteen years old and has never consumed alcohol. This adolescent is suffering from nonalcoholic fatty liver disease (NAFLD).

The liver is not an isolated organ; it works in conjunction with almost every other system of the body, including the digestive, endocrine, and circulatory system. For instance, the liver regulates blood glucose levels, which is the body's primary source of energy and fat. The liver metabolizes, *i.e.* synthesizes and breaks down glucose and fat, depending on the body's needs, and stores the excess energy temporarily in the form of glycogen and triglycerides (Manco, 2011). In the case of alcohol-induced fatty liver, alcohol disrupts the fat metabolism, causing an influx and accumulation of free fatty acids in the liver (Orman et al., 2013). NAFLD causes the same effect, driven primarily by obesity (Nobili et al., 2009).

In fact, NAFLD prevalence rates parallel the so-called "globesity" (global obesity) rates (Corte et al., 2012). In the United States alone, 17% of children are considered overweight and obese by the Centers for Disease Control and Prevention (CDC); of these, 52% have NAFLD (Oddy et al., 2013). The proportion of children

suffering from this disease represents 3%–13% of the total US adolescent population. NAFLD is defined by Anderson et al. (2015) as "the accumulation of fat in liver in the absence of excessive alcohol consumption or other known liver pathologies" (p. 2). The first adult case was reported in 1980 by Ludwig, and in children by Moran in 1983 (Bozic et al., 2013). From 1994 to 2004, its prevalence rate increased to 62.84%, and within four years, it rose to 75.1% (Younossi et al., 2011). In 2015, it is currently the leading cause of liver disease in children (Koot et al., 2015), with patients as young as three years of age (Manco et al., 2008).

Since NAFLD is more common amongst Hispanic American than African American populations, 45% versus 24% respectively, there may be a strong genetic predisposition toward developing the disease (Marzuillo et al., 2014). However, besides genetics, other factors can also significantly contribute to the progression of the disease. These are explained in terms of a double hit hypothesis, as shown in Figure 1. The first hit refers to the accumulation of fat in liver cells. This sensitizes the liver to other factors, known collectively as the second hit, which aggravate the liver further, leading to inflammation, scarring, and if left untreated, even cancer (Veena et al., 2014). The majority of pediatric NAFLD patients also show psychological symptoms (St-Jules et al., 2013), reporting a significantly lower quality of life compared to healthy children, 72% versus 83% respectively. Their symptoms include fatigue, depression, insomnia, and poor school performance (Kistler et al., 2010). Children with NAFLD also have a thirteen-fold increased risk of death or of requiring a liver transplant as compared to their healthy counterparts (Feldstein et al., 2009). Undoubtedly, we are letting one preventable and reversible global epidemic, obesity, exacerbate the spread of another preventable disease, advancing both to chronic and fatal conditions. This article examines how NAFLD develops in children and explores preventative measures and treatment options, including lifestyle modifications, in order to present the best interventions for combatting the rapidly increasing rate of pediatric NAFLD.

CHILDREN WITH NAFLD ALSO HAVE A THIRTEEN-FOLD INCREASED RISK OF DEATH OR OF REQUIRING A LIVER TRANSPLANT

CONTEXTUAL FACTORS THAT FAVOR NAFLD AND THE TWO-HIT HYPOTHESIS

Given that children have been exposed to an unhealthy lifestyle for a much shorter period than adults, genetics may play more of a dominant role in pediatric NAFLD prevalence than adult NAFLD; especially considering that in some cases, children have the disease despite optimal dietary and lifestyle habits (Nobili et al., 2014). In fact, one study found that the immediate family members of children with NAFLD had a much higher percentage of liver fat content, with siblings at 9.3% and parents at 14% fat, than the family members of the children without the disease, whose siblings were at 2.7% and parents at 7% (Schwimmer et al., 2009). It is evident that NAFLD is impacted by an individual's genetics; current research has identified multiple genes that play a role in its development (Dongiovanni et al., 2013).

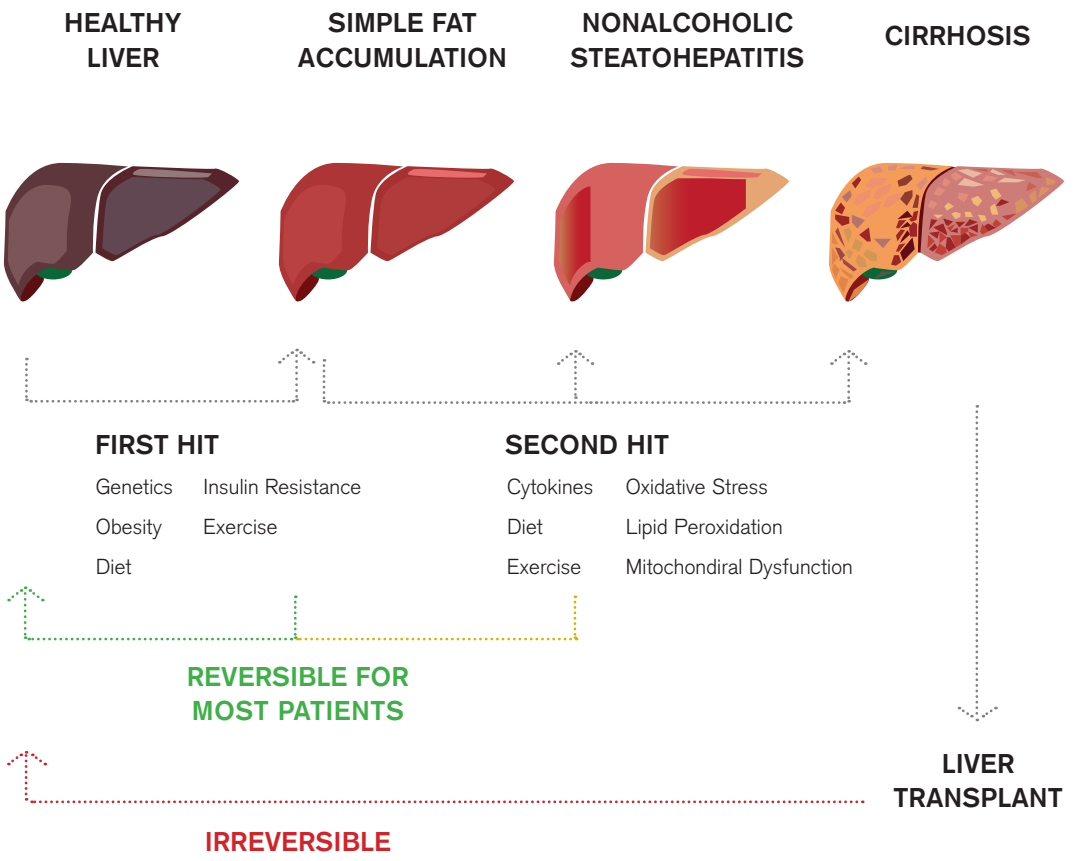


FIGURE 1. The Progression of NAFLD

NAFLD does not refer to one particular disease with a set of symptoms. Rather, it is a spectrum of diseases, with three key stages. The earliest and most prevalent stage of NAFLD is called simple steatosis (Ozgur et al., 2013). This stage involves the accumulation of triglycerides in the liver cells due to an imbalance between the import and export of fatty acids (Nobili et al., 2009), without significant liver inflammation (Roberts, 2007). While simple steatosis is asymptomatic and reversible (Rafeey et al., 2009), if no action is taken to revert the liver's condition, simple steatosis progresses to nonalcoholic steatohepatitis (NASH). At this stage, liver cells contain harmful amount of fat molecules, and there is significant inflammation with damaged and dead liver cells, which causes scarring (Aqel & Dibaise, 2015). If the liver cells do not regenerate at the same rate as they die, further scarring occurs, leading to cirrhosis (Ozgur et al., 2013), and in some cases, cancer (Boursier & Diehl, 2015). Unlike simple steatosis, NASH is not asymptomatic, although it may be reversible in some cases. However, once the disease has progressed to cirrhosis, the damage is irreversible and fatal (Rafeey et al., 2009).

The most apparent and globally conspicuous genetic relationship of NAFLD is with the gene called PNPLA3, patatin-like phospholipase domain-containing protein 3 (Lim et al., 2010), which is used to make an enzyme called adiponutrin (Park et al., 2015). Adiponutrin is primarily synthesized in liver and adipose (fat) tissue. In a healthy individual, it acts as a triglyceride synthase in adipose cells, and as a triglyceride hydrolase in liver cells. This means it can both synthesize and break down triglycerides, depending on the cell and the environmental factors that it is exposed to (Park et al., 2015). In a NAFLD patient, a single nucleotide mutation, in which cytosine is substituted with guanosine, creates a different version of PNPLA3, a variant called rs738409-G allele, which codes for a modified adiponutrin protein that lacks its hydrolytic function (Marzuillo et al., 2014). This variant is correlated with a diet consisting of highly sweetened beverages and excess carbohydrates (Nobili et al., 2014). This adiponutrin variant continues to synthesize triglycerides in fat tissue, as it would in any healthy individual; however, it does not effectively break down proliferating triglycerides in the liver (Park et al., 2015). The accumulation of these fat particles causes severe steatosis, inflammation, and fibrosis of the liver (Valenti et al., 2010). The PNPLA3-G variant and modified adiponutrin are most commonly found among the Hispanic population, which is also the population with the highest NAFLD prevalence (Marzuillo et al., 2014). Conversely, the population with the lowest rate of NAFLD (African Americans) have been found to carry a different variant of PNPLA3, called rs6006460-T-allele, which is associated with low hepatic fat (Pan & Fallon, 2014).

Marzuillo has hypothesized that there are also relationships between NAFLD and other genes, such as the glucokinase regulatory (GCKR) gene. The GCKR gene codes for a protein called glucokinase regulatory protein. This protein is responsible for mediating a liver enzyme called glucokinase (GCK), which carries out glucose and fat metabolism (Tan et al., 2013). In order to prevent synthesis of excess fatty acids, glucokinase regulatory protein binds to GCK and inhibits its activity. An

Free Fatty Acids
Long chains of carbon atoms that are derived from larger fat molecules

Gene
A piece of information on DNA that results in a specific trait

Hyperlipidemia
A condition in which there are high levels of fat molecules (lipids) in the blood

Lipid Peroxidation
A process in which molecules with oxygen (known as free oxygen radicals) "steal" electrons from lipids, preventing their proper functioning

Mutation
A permanent change in a part of the DNA

Nucleotides
Organic molecules that make up the DNA

Oxidation
Loss of electrons from a molecule or atom

Trait
A physiological characteristic of an organism that may be inherited

Triglycerides
Specific type of fat molecule, derived from glycerol, and three fatty acids

Fibrosis
Formation of excess fibrous connective tissue in an organ; scarring

Glucose
A six carbon sugar that acts as a primary source of energy for the body

Glycogen
A large sugar made up of multiple glucose molecules; this acts as the storage form of glucose/energy

Hyperinsulinemia
A condition in which the levels of insulin in the blood are relatively higher than glucose levels

NAFLD patient, however, has a different version of the GCKR gene, called rs780094 allele. This variant produces a dysfunctional GCKR, which is unable to inhibit GCK. Consequently, GCK continues to synthesize triglycerides, which accumulate in the liver cells (Petta et al., 2014). The prevalence of both the PNPLA3 and GCKR variants are lowest in the African American population. By contrast, GCKR variant is more prevalent in Han Chinese as compared to high PNPLA3 in Hispanic Americans (Lin et al., 2014).

While such genes predispose an individual to NAFLD, the development of the disease can be described in terms of a *two-hit hypothesis*. The first hit refers to the factors that lead to the accumulation of fat molecules in the liver, resulting in NAFLD (Nanda, 2004); specifically, obesity, insulin resistance, and hyperinsulinemia (Marzuillo et al., 2015). These factors, along with others like hypertension and glucose regulation, are categorized under metabolic syndrome (Alkhater, 2015). MtS, or metabolic syndrome, is an umbrella term used for a group of risk factors that lead to the development of cardiovascular diseases and type 2 diabetes mellitus (Schwimmer et al., 2008). Considering NAFLD exacerbates the progression of cardiovascular diseases, it is believed to be the hepatic manifestation of metabolic syndrome (Alkhater, 2015). While all children with NAFLD have at least one other disease that is categorized under MtS, obese adolescents are at five times the risk of the syndrome. This link between NAFLD and metabolic syndrome develops in early adolescence due to obesity-driven insulin resistance (Manco, 2011).

Insulin resistance is defined as the body's inability to lower blood glucose levels in the fasting state, despite the rise in insulin concentration in the blood (Mann et al., 2015). In a healthy body, insulin triggers the muscle, fat, and liver cells to uptake glucose from the bloodstream and either metabolize it for energy or store it as glycogen. Simultaneously, it suppresses the production of free fatty acids in both adipose and liver tissue (Utzschneider & Kahn, 2006). The switch between metabolizing fat or glucose depends on the availability and demand of each macronutrient. In insulin-resistant patients, however, the body loses its ability to make that shift. Neither the adipose tissue nor the liver respond to insulin, glucose does not get absorbed by the cells (Lee et al., 2015), and free fatty acid synthesis continues in adipose and liver cells. Moreover, the adipose cells fail to expand in order to store the excess fat, which consequently gets delivered to the liver (Manco, 2011). Much like NAFLD and metabolic syndrome, fat accumulation and insulin resistance are a cyclical process. That is, the hepatic fat (Marzuillo et al., 2015), as well as the increased rate of triglyceride synthesis (Giorgio et al., 2013), exacerbates insulin

resistance by preventing the activation of insulin receptors (Berardis & Sokal, 2014), worsening hepatic steatosis.

Insulin sensitivity is 55% lower among obese adolescents with NAFLD as compared to healthy children (Lee et al., 2015). Indeed, obesity is a major risk factor for NAFLD (Alkhater, 2015). Studies have shown that while 2–6% of the pediatric population has been diagnosed with the disease, this value significantly increases to 20–50% for obese children. It is especially prevalent in pubertal children and adolescents than in pre-pubertal children (Ackam et al., 2013). At the onset of puberty, children go through hormonal imbalances, fat redistribution throughout the body, and a decrease in insulin sensitivity. Although most of these changes subside as a part of the maturation process (Cruz et al., 2005), the increase in blood insulin levels is sustained throughout adolescence (Loomba et al, 2009). Obesity, insulin levels, and triglyceride synthesis are connected in a cyclical process; an increase in one aggravates another.

These first hit factors then branch into different pathologies, affecting the body as a whole. With proper treatment, the condition is reversible (Lim et al., 2010). The second hit, however, can perpetuate NAFLD and cause further liver damage. This can lead to nonalcoholic steatohepatitis (NASH), which makes reversal of the condition more difficult (Marzuillo et al., 2015). The second hit involves lipid peroxidation and inflammatory cytokine activation, both of which are related to mitochondrial dysfunction (Giorgio et al., 2013). Liver cells are rich in mitochondria, which are cellular organelles that use free fatty acids and oxygen to synthesize energy, carbon dioxide, and water (Basaranoglu et al., 2013). A dysfunctional mitochondrion, however, is unable to efficiently convert most of the oxygen into the required products, instead creating molecules known as reactive oxygen species, or ROS (Nanda, 2004). These ROS negatively affect many cellular processes. They carry out lipid peroxidation (Mylonas & Kouretas, 1999), oxidizing—and thus damaging—fatty acids (necessary for important cellular processes such as intracellular cell signaling and storage). When lipid peroxidation occurs at a high rate, oxidized fatty acids are synthesized at a toxic level. The damage from these molecules overwhelms the repair capacity of the cell, contributing to the fat stored in the liver cells. Moreover, in another cyclic process, lipid peroxidation causes further mitochondrial dysfunction, resulting in overproduction of oxygen species (Takahashi et al., 2010).

ROS also cause an imbalance in cytokine production (Marzuillo et al., 2015) by triggering the release of inflammatory molecules from ectopic fatty tissue (Fitzpatrick et al, 2012), while simultaneously inhibiting the release of anti-inflammatory molecules (Berardis & Sokal, 2014). These inflammatory cytokines include tumor necrosis factor-alpha (TNF-α) and leptin. TNF-α not only enhances insulin resistance (Roberts, 2007), but also activates a protein which binds to the DNA to induce the production of more ROS (Veena et al.,

Adipose Tissue
Body fat, found under the skin

Cytokine
Proteins responsible for cellular signaling. They are an active part of the immune system and regulate the maturation, growth, and responsiveness of certain cells

Hepatic Fat
Liver fat

Insulin
A hormone produced and secreted by pancreas to regulate the synthesis and breakdown of carbohydrates and fats

2014). Given a high concentration of ROS, TNF-α induces liver inflammation, liver cell death, and fibrosis (Manco et al., 2007). Along with TNF-α, leptin synthesis is also high in obese patients with NAFLD. Leptin is a satiety cytokine, which the brain fails to respond to in cases of chronic obesity (Giorgio et al., 2013). Insensitivity to leptin prevents an individual from recognizing when they are full, and so they continue to eat and gain weight. Note that the damages caused by ROS—the oxidation of lipids, decrease in ATP production, and increase in inflammatory cytokine release—are together categorized under the term oxidative stress. It is due to the collective effects of these events that the liver cells die (Ayala, 2014), thus causing inflammation and scarring of the organ (Basaranoglu et al., 2013).

Along with the aforementioned physiological changes, adolescents also gain increasing freedom to make their own decisions, including those about diet and physical activity. For many, these decisions result in switching to a diet of high calorie foods and a sedentary lifestyle (Giorgio et al., 2013). Therefore, it is important to recognize the type of diet that promotes NAFLD progression. Children with NAFLD tend to eat a diet higher in total saturated fat and refined sugars as compared to both obese and lean children who do not have NAFLD (Mitchel & Lavine, 2014). Fructose, in particular, is associated with NAFLD, as patients with a higher intake of sugar-sweetened beverages are at an increased risk of developing the condition regardless of their age, sex, and BMI (Veena et al., 2014). Fructose is primarily metabolized in the liver, through a mechanism very similar to that of ethanol (Lim et al., 2010), which increases lipid synthesis. It also acts as a pro-inflammatory mediator of NAFLD, contributing to liver inflammation (Alkhater, 2015).

Fructose can also induce bacterial overgrowth and increased bacterial permeability in the small intestine, causing the movement of bacteria from the gut into the blood circulation (Michail et al., 2015). This increase in permeability leads to bacterial toxicity and the conversion of liver cells to myofibroblasts, which release pro-inflammatory molecules such as TNF-α (Alkhater, 2015). The toxins from the gut bacteria also activate the complement system (Zhan et al., 2010), further exposing liver to inflammation (Lim et al., 2010). One of the main bacterial products involved in NAFLD is called lipopolysaccharide (LPS). As an active component of an endotoxin, LPS triggers a cascade of several enzymes involved in the inflammatory pathway (Aqel & DiBaise, 2015).

PREVENTATIVE ACTIONS AND TREATMENT FOR IMPROVING CHILD HEALTH

Before treatment can begin, a diagnosis must be made. According to many studies, this is a fairly difficult process primarily because the early stages of NAFLD are either asymptomatic or the symptoms are unrelated to the liver (Nanda, 2004). There is also a general lack of non-invasive diagnostic tests (Anderson et al., 2015). A physical examination is of no help as it results in a diagnosis of obesity (Roberts, 2007). Consequently, NAFLD diagnosis is based on either elevated levels of aminotransferase

Ectopic Fat
The fat that accumulates in organs other than adipose tissue, *e.g.* in the liver, muscle tissues, pancreas and arteries

Fructose
Fruit sugar made up of molecules of glucose and sucrose

Intracellular Cell Signaling
Part of a communication system in the body that activates and coordinates cell actions

(ALT), an enzyme that is secreted by damaged liver cells, or by decreased levels of adiponectin. However, even these results are not definitive diagnostic criteria. In fact, there is no proper threshold of ALT with respect to age and sex to indicate NAFLD (Anderson et al., 2015). The only sure way to diagnose NAFLD is to first disprove all other possibilities (Hourigan et al., 2015), then carry out a liver biopsy, especially to distinguish between simple steatosis and NASH (Roberts, 2007).

Once diagnosed, treatment for pediatric NAFLD must occur as soon as possible, with a focus on reversing steatosis and promoting healthy growth and development (Roberts, 2007). The best way known is through weight loss, which decreases the concentration of free fatty acids in liver, increases insulin sensitivity by metabolizing glucose rather than fat, and reduces the synthesis of ROS (Mitchel & Lavine, 2014). Thus, dietary changes and adequate exercise are considered the first line of defense against progression of NAFLD to NASH (Nobili et al., 2009). A low glycemic index (GI) diet is appropriate for weight loss (Loomba et al., 2009), consisting of 50–60% carbohydrates, 23–30% fats, and 15–20% proteins. The intake of high fructose beverages must be reduced as greater amounts of sugar stimulate synthesis of fatty acids in the body, proliferating the fat in the liver (Manco et al., 2008). Since excess fructose is also associated with insulin resistance, steatosis, and oxidative stress, decreasing its consumption will significantly improve liver health (Manco, 2011). Changing to a healthier diet can also significantly decrease the blood levels of ALT (Nobili et al., 2009). Some studies have recorded lower ALT and triglyceride levels after a twelve-month treatment with an omega-3 fatty acid DHA (Berardis & Sokal, 2014). Omega fatty acids, such as DHA and EPA, have been shown to reduce fat deposits by activating certain cellular pathways that induce breakdown of fatty acids and inhibit their synthesis (Manco et al., 2008).

Physicians and researchers agree that proper diet is significant in reversing NAFLD in pediatric patients. However, it is important to cater the lifestyle modifications to each individual; this serves several purposes. First, it improves compliance, which may be a zchallenge when working with children and adolescent patients. Second, dietary and physical requirements must be continuously reevaluated to account for the child's developmental rate, changes in body weight, height, etc. (St. Jules et al., 2013). It is extremely important that the child's needs for growth are met (Berardis & Sokal, 2014), especially during puberty. Physical changes should not be the only aspect accorded importance. During this period, most children and adolescents are already sensitive about their body image and may have low self-esteem. Therefore, overemphasizing the need for weight reduction should be avoided to prevent negative psychological effects (St. Jules et al., 2013). It is in fact harmful to lose a great amount of weight in a short period of time, as this may increase liver injury (Nobili et al., 2009). In order to ensure gradual weight loss, children should begin with moderate exercise like brisk

Complement System
Consists of small proteins that are synthesized in the liver, which complement antibodies and phagocytes in the immune system

walking or swimming (Veena et al., 2014). Although forty-five minutes of aerobic exercise per day is recommended (Nobili et al., 2009) for treatment and as positive reinforcement (Roberts, 2007), more studies are required to determine the benefits of exercise on its own, as well as with dietary changes (Deldin & Lee, 2013).

Further research is also required in the pharmacological field, since there is no drug treatment approved specifically for NAFLD (Veena et al., 2014). Pediatric NAFLD treatments are especially difficult because they must account for several factors, such as growth and development, hormonal changes, and rapid lifestyle modifications. Any medication prescribed must be unresponsive towards the changes in an adolescent body (Berardis & Sokal, 2014). For now, given that NAFLD progresses due to the cyclical process involving obesity, diabetes, insulin resistance, and oxidative stress, those are the pathologies targeted by most medicines (Nanda, 2004).

Bile
A fluid produced by the liver that is discharged into the small intestine to assist in the digestion of ingested fats

Noninvasive
Refers to treatment methods that do not require incisions, and lessen pain, healing time, and risk of infection

Steatosis
Refers to accumulation of lipids inside a cell

The only weight loss medication approved by the FDA for pediatric use is Orlistat. Although this drug inhibits excess fat absorption, it also interferes with absorption of fat-soluble vitamins. Nonetheless, it may reverse steatosis and insulin resistance (Mitchel & Lavine, 2014). Another medication, metformin, is well documented as a safe and effective treatment for diabetes (Loomba et al, 2009) and insulin resistance (Nanda, 2004), and so is often prescribed to target that aspect of NAFLD. However, many studies have shown that although metformin helps to decrease insulin resistance, it does not improve liver condition any more than lifestyle changes (Manco, 2011). Another class of insulin sensitizers called thiazolidinediones have been shown to improve insulin sensitivity and reverse steatosis in adults (Nanda, 2004), but are not yet prescribed to children due to the lack of scientific support as well as the known side effects, which include weight gain, cardiovascular disease, and heart failure (Alkhater, 2015).

Antioxidants such as vitamin E supplements are also considered to be a possible treatment option. In some cases, supplements have been shown to decrease the ballooning of the liver cells in NASH (Berardis & Sokal, 2014). However, much like insulin sensitizers, there are either not enough cohort studies on their long term effects (Veena et al., 2014), or the few studies that have been done show no sign that vitamin E is more effective than lifestyle changes (Sarkhy et al., 2014). On the other hand, if vitamin E is administered with UDCA, an acid that deactivates bile to prevent the killing of liver cells, in conjunction with proper diet and exercise, ALT levels show improvement (Cho et al., 2012). In comparison, DHA, an omega-3-fatty acid, results in decreases in both the ALT and triglyceride levels (Alkhater, 2015). Probiotics have been found to have a similar effect (Berardis & Sokal, 2014); in fact, probiotics are being considered as highly valuable anti-inflammatory agents that work by decreasing bacterial translocation (Mitchel & Lavine, 2014).

In extreme chronic cases, and as a last resort, surgery may be considered. Bariatric surgery and transplant is seen as

a treatment option for children whose lifestyle changes were unsuccessful or for those who were diagnosed in the chronic stage. There are not enough studies on the long term safety of pediatric bariatric surgery and its effects on hepatic steatosis and inflammation. However, it has been shown to improve ALT levels (Alkhater, 2015). There are two types of surgeries, gastric banding and Roux-en-Y gastric bypass. Despite variable long-term results, banding is believed to have the least amount of risk and is reversible. The gastric bypass procedure is believed to be more efficient, though it may lead to nutrient deficiencies and protein malnutrition (Mitchel & Lavine, 2014).

Transplantation in NAFLD patients is often complicated by the conditions that come with the disease such as obesity, diabetes, and hyperlipidemia (Nanda, 2004). A condition called post-transplant metabolic syndrome is seen in some children who undergo liver transplants. Moreover, the immunosuppressive therapy that follows a transplant can lead to diabetes, hypertensions, cardiovascular diseases, and steatosis (Nobili & de Ville de Goyet, 2013). Prospective donors, especially those at risk for developing NAFLD themselves, must first undergo liver biopsies (Mathur et al., 2007).

CONCLUSION

Nonalcoholic fatty liver disease (NAFLD) is an increasingly prevalent condition that interferes with the proper growth and development of children. It has a wide array of causative factors, and its effects extend beyond the liver to the whole body. There is a need for discussions about NAFLD at the public level, as fatty liver is mistakenly only associated with alcohol consumption. Obesity, heart disease, and hypertension are rarely associated with the liver, despite being strong contributors to the progression of NAFLD. Since it is imperative that children with NAFLD be diagnosed as soon as possible, increasing knowledge on the disease will allow more cases to be caught in time. In fact, all children—especially those at risk of obesity, and those with a family history of fatty liver—should be screened for NAFLD on a regular basis. This highlights the dire need for further research to develop new diagnostic testing that is noninvasive, accurate, and less expensive.

More long-term research is also needed to determine the efficacy of specific diets, supplements, and medications that are seen as options for NAFLD treatment. Currently, it is extremely important for NAFLD patients to be served by a multidisciplinary healthcare team. Dieticians, hepatologists, psychologists, and cardiologists need to evaluate the cases and be involved with the patient (Nobili et al., 2009). For pediatric patients, family involvement is also necessary to maintain nutritional knowledge and implement physical activity—along with clinical treatment.

Hepatologists
Individuals who study the liver and its pathologies

REFERENCES

Akcam, M., Boyaci, A., Pirgon, O., Koroglu, M., & Dundar, B.N. (2013). Importance of the Liver Ultrasound Scores in Pubertal Obese Children with Nonalcoholic Fatty Liver Disease. *Clinical Imaging*, 37(3), 504–8. http://dx.doi.org/10.1016/j.clinimag.2012.07.011

AlKhater, S. A. (2015). Paediatric Non-Alcoholic Fatty Liver Disease: An Overview. *Obesity Reviews*, 16(5), 393–405. http://dx.doi.org/10.1111/obr.12271.

Anderson, E.L., Howe, L.D., Jones, H.E., Higgins, J.P.T., Lawlor, D.A., & Fraser, A. (2015). The Prevalence of Non-Alcoholic Fatty Liver Disease in Children and Adolescents: A Systematic Review and Meta-Analysis. V. Wong (Ed.), *PLOS ONE, 10(10),* e0140908/1–14. http://dx.doi.org/10.1371/journal.pone.0140908

Aqel, B., & DiBaise J.K. (2015). Role of the Gut Microbiome in Nonalcoholic Fatty Liver Disease. *Nutrition in Clinical Practice*, 30(6), 780–86. http://dx.doi.org/10.1177/0884533615605811

Ayala, A., Muñoz, M.F., & Argüelles, S. (2014). Lipid Peroxidation: Production, Metabolism, and Signaling Mechanisms of Malondialdehyde and 4-Hydroxy-2-Nonenal. *Oxidative Medicine and Cellular Longevity*, 2014, 1–31. http://dx.doi.org/10.1155/2014/360438

Basaranoglu, M., Basaranoglu, G., & Sentürk, H. (2013). From fatty liver to fibrosis: A tale of "second hit." *World Journal of Gastroenterology*, 19(8), 1158–1165. http://dx.doi.org/10.3748/wjg.v19.i8.1158

Berardis, S., & Sokal, E. (2014). Pediatric Non-Alcoholic Fatty Liver Disease: An Increasing Public Health Issue. *European Journal of Pediatrics*, 173(2), 131–39. http://dx.doi.org/10.1007/s00431-013-2157-6

Bozic, M. A., Subbarao, G., & Molleston, J.P. (2013). Pediatric Nonalcoholic Fatty Liver Disease. *Nutrition in Clinical Practice*, 28(4), 448–58. http://dx.doi.org/10.1177/0884533613489153

Cho, T., Kim, J.Y., & Paik, S.S. (2012). The Efficacy of Pharmacological Treatment in Pediatric Nonalcoholic Fatty Liver Disease. *Pediatric Gastroenterology, Hepatology & Nutrition* 15(4), 256–265. http://dx.doi.org/10.5223/pghn.2012.15.4.256

Corte, C., Alisi, A., Saccari, A., De Vito, R., Vania, A., Valerio, N. (2012). Nonalcoholic Fatty Liver in Children and Adolescents: An Overview. Journal of Adolescent Health, 51(4), 305–312. http://dx.doi.org/10.1016/j.jadohealth.2012.01.010

Cruz, M.L., Shaibi G.Q., Weigensberg, M.J., Spruijt-Metz. D., Ball, G.D.C., & Goran, M.I. (2005). PEDIATRIC OBESITY AND INSULIN RESISTANCE: Chronic Disease Risk and Implications for Treatment and Prevention Beyond Body Weight Modification. *Annual Review of Nutrition*, 25(1), 435–68. http://dx.doi.org/10.1146/annurev.nutr.25.050304.092625

Deldin, A.R., & Lee S. (2013). Role of Physical Activity in the Treatment of Nonalcoholic Fatty Liver Disease in Children and Adolescents. Applied Physiology, Nutrition, and Metabolism, 38(8), 805–12. http://dx.doi.org/10.1139/apnm-2012-0503

Dongiovanni, P., Anstee, Q., & Valenti, L. (2013). Genetic Predisposition in NAFLD and NASH: Impact on Severity of Liver Disease and Response to Treatment. *Current Pharmaceutical Design 19*(29), 5219–38. http://dx.doi.org/10.2174/13816128113199990381

Feldstein, A.E., Charatcharoenwitthaya, P., Treeprasertsuk, S., Benson, J.T., Enders, F.B., & Angulo, P. (2009). The natural history of non-alcoholic fatty liver disease in children: a follow-up study for up to 20 years. *Gut, 58*(11), 1538–44. http://dx.doi.org/10.1136/gut.2008.171280Fitzpatrick, E., Dew, T.K., Quaglia, A., Sherwood, R.A., Mitry, R.R., & Dhawan, A. (2012). Analysis of adipokine concentrations in paediatric non-alcoholic fatty liver disease: adipokines in paediatric NAFLD. *Pediatric Obesity, 7*(6), 471–79. http://dx.doi.org/10.1111/j.2047-6310.2012.00082.x

Giorgio, V., Prono, F., Graziano, F., & Nobili, V. (2013). Pediatric nonalcoholic fatty liver disease: old and new concepts on development, progression, metabolic insight and potential treatment targets. *BMC Pediatrics,* 13(1), 1. http://dx.doi.org/ 10.1186/1471-2431-13-40

Hourigan, S. K., Torbenson, M., Tibesar, E., & Scheimann, A. O. (2015). The full spectrum of hepatic steatosis in children. *Clinical Pediatrics*, 54(7), 635–642. http://doi.org/10.1177/0009922814566927

Kistler, K. D., Molleston, J., Unalp, A., Abrams, S. H., Behling, C., Schwimmer, J. B., & for the Nonalcoholic Steatohepatitis Clinical Research Network (NASH CRN). (2010). Symptoms and quality of life in obese children and adolescents with non-alcoholic fatty liver disease. *Alimentary Pharmacology & Therapeutics, 31*(3), 396–406. http://doi.org/10.1111/j.1365-2036.2009.04181.x

Koot, B.G. P., de Groot, E., van der Baan-Slootweg, O.H., Bohte, A.E., Nederveen, A.J., Jansen, P.L.M., Stoker, J., & Benninga, M.A. (2015). Nonalcoholic fatty liver disease and cardiovascular risk in children with obesity. *Obesity, 23*(6), 1239–43. http://dx.doi.org/10.1002/oby.21076

Lee, S., Rivera-Vega, M., Alsayed, H. M. A. A., Boesch, C. and Libman, I. (2015). Metabolic inflexibility and insulin resistance in obese adolescents with non-alcoholic fatty liver disease. *Pediatric Diabetes* 16, no. 3 (May 2015): 211–18. http://dx.doi.org/10.1111/pedi.12141

Lim, J. S., Mietus-Snyder, M., Valente, A., Schwarz, J.M., & Lustig, R.H. (2010). The Role of Fructose in the Pathogenesis of NAFLD and the Metabolic Syndrome. Nature Reviews Gastroenterology & Hepatology, 7(5), 251–64. http://dx.doi.org/10.1038/nrgastro.2010.41

Lin, Y.-C., Chang, P.-F., Chang, M.-H., & Ni, Y.-H. (2014). Genetic variants in GCKR and PNPLA3 confer susceptibility to nonalcoholic fatty liver disease in obese individuals. *The American Journal of Clinical Nutrition, 99*(4), 869–874. http://doi.org/10.3945/ajcn.113.079749

Loomba, R., Sirlin, C. B., Schwimmer, J. B., & Lavine, J. E. (2009). Advances in pediatric nonalcoholic fatty liver disease. *Hepatology, 50*(4), 1282–1293. http://doi.org/10.1002/hep.23119

Manco, M., Marcellini, M., Giannone, G., & Nobili, V. (2007). Correlation of Serum TNF-α Levels and Histologic Liver Injury Scores in Pediatric Nonalcoholic Fatty Liver Disease. *American Journal of Clinical Pathology, 127*(6), 954–960. http://doi.org/10.1309/6VJ4DWGYDU0XYJ8Q

Manco, M., Bottazzo, G.F., DeVito, R., Marcellini M., Mingrone, G., & Nobili, V. (2008). Nonalcoholic Fatty Liver Disease in Children. *Journal of the American College of Nutrition, 27*(6), 667–76. http://dx.doi.org/10.1080/07315724.2008.10719744

Manco, M. (2010). Pediatric Insulin Resistance and NAFLD. *Journal of the American College of Nutrition, 29*(4), 435–435. http://doi.org/10.1080/07315724.2010.10719865

Manco, M. (2011). Metabolic Syndrome in Childhood from Impaired Carbohydrate Metabolism to Nonalcoholic Fatty Liver Disease. *Journal of the American College of Nutrition, 30*(5), 295–303. http://doi.org/10.1080/07315724.2011.10719972

Mann, J. P., Goonetilleke, R., & McKiernan,P. (2015). Paediatric Non-Alcoholic Fatty Liver Disease: A Practical Overview for Non-Specialists. *Archives of Disease in Childhood, 100*(7), 673–77. http://dx.doi.org/10.1136/archdischild-2014-307985

Marzuillo, P., Grandone, A., Perrone, L., & del Giudice , E.M. (2015). Understanding the pathophysiological mechanisms in the pediatric non-alcoholic fatty liver disease: The role of genetics. *World Journal of Hepatology, 7*(11), 1439–43. http://doi.org/10.4254/wjh.v7.i11.1439

Marzuillo, P., del Giudice , E.M., & Santoro, N. (2014). Pediatric fatty liver disease: Role of ethnicity and genetics. *World Journal of Gastroenterology, 20*(23), 7347–55. http://doi.org/10.3748/wjg.v20.i23.7347

Mathur, P., Das, M. K., & Arora, D. P. N. K. (2007). Non-alcoholic fatty liver disease and childhood obesity. *The Indian Journal of Pediatrics, 74*(4), 401–408. http://doi.org/10.1007/s12098-007-0068-0

Michail, S., Lin, M., Frey, M. R., Fanter, R., Paliy, O., Hilbush, B., & Reo, N. V. (2015). Altered gut microbial energy and metabolism in children with non-alcoholic fatty liver disease. *FEMS Microbiology Ecology, 91*(2), 1–9. http://doi.org/10.1093/femsec/fiu002

Mitchel, E. B., & Lavine, J. E. (2014). Review article: the management of paediatric nonalcoholic fatty liver disease. *Alimentary Pharmacology & Therapeutics, 40*(10), 1155–1170. http://doi.org/10.1111/apt.12972

Mylonas, C., & D. Kouretas. D. (1999). *Lipid Peroxidation and Tissue Damage. In Vivo, 13*(3), 295–309.

Nanda, K. (2004). Non-alcoholic steatohepatitis in children. *Pediatric Transplantation, 8*(6), 613–618. http://doi.org/10.1111/j.1399-3046.2004.-00241.x

Nobili, V., Alisi, A., & Raponi, M. (2009). Pediatric Non-Alcoholic Fatty Liver Disease: Preventive and Therapeutic Value of Lifestyle Intervention. *World Journal of Gastroenterology, 15*(48), 6017-22. http//dx.doi.org/10.3748/wjg.15.6017

Nobili, V., Parola, M., Alisi, A., Marra, F., Piemote, F., Mombello, C., Sutti, S., Povero, D., Maina, V., Novo, E., & Albano, E. (2010). Oxidative stress parameters in paediatric non-alcoholic fatty liver disease. *International Journal of Molecular Medicine, 26*(4), 471–476. http://doi.org/10.3892/ijmm_00000487

Nobili, V., Cutrera, R., Liccardo, D., Pavone, M., Devito, R., Giorgio, V., Verrillo, E., Baviera, G., & Musso, G. (2013). Obstructive Sleep Apnea Syndrome Affects Liver Histology and Inflammatory Cell Activation in Pediatric Nonalcoholic Fatty Liver Disease, Regardless of Obesity/Insulin Resistance. *American Journal of Respiratory and Critical Care Medicine, 189*(1), 66–76. http://doi.org/10.1164/rccm.201307-1339OC

Nobili, V., & de Ville de Goyet, J. (2013). Pediatric post-transplant metabolic syndrome: New clouds on the horizon. *Pediatric Transplantation, 17*(3), 216–223. http://doi.org/10.1111/petr.12065

Nobili, V., Carpino, G., Alisi, A., Vito, R. D., Franchitto, A., Alpini, G., Onori, P., & Gaudio, E. (2014). Role of Docosahexaenoic Acid Treatment in Improving Liver Histology in Pediatric Nonalcoholic Fatty Liver Disease. F.M. Sladek (Ed.), *PLOS ONE, 9*(2), e88005. http://doi.org/10.1371/journal.pone.0088005

Nobili, V., Liccardo, D., Bedogni, G., Salvatori, G., Gnani, D., Bersani, I., Alisi, A., Valenti, L., & Raponi, M. (2014). Influence of dietary pattern, physical activity, and I148M PNPLA3 on steatosis severity in at-risk adolescents. *Genes & Nutrition, 9*(3), 1–7. http://doi.org/10.1007/s12263-014-0392-8

Oddy, W. H., Herbison, C. E., Jacoby, P., Ambrosini, G. L., O'Sullivan, T. A., Ayonrinde, O. T., Olynyk, J.K., et al. (2013). The Western Dietary Pattern Is Prospectively Associated With Nonalcoholic Fatty Liver Disease in Adolescence. *The American Journal of Gastroenterology, 108*(5), 778–785. http://doi.org/10.1038/ajg.2013.95

Orman, E.S., Odena, G., & Bataller, R. (2013). Alcoholic Liver Disease: Pathogenesis, Management, and Novel Targets for Therapy: Alcoholic Liver Disease. *Journal of Gastroenterology and Hepatology, 28*(2), 77–84. http://dx.doi.org/10.1111/jgh.12030

Özgür, P., Hüseyin, B., Ferhat, C., Hüseyin K., & Nuri, D.B. (2013). Association Between Insulin Resistance and Oxidative Stress Parameters in Obese Adolescents with Non-Alcoholic Fatty Liver Disease. *Journal of Clinical Research in Pediatric Endocrinology, 5*(1), 33–39. http://dx.doi.org/10.4274/Jcrpe.825

Pan, J.-J., & Fallon, M. B. (2014). Gender and racial differences in nonalcoholic fatty liver disease. *World Journal of Hepatology, 6*(5), 274–283. http://doi.org/10.4254/wjh.v6.i5.274

Park, J.-H., Cho, B., Kwon, H., Prilutsky, D., Yun, J. M., Choi, H. C., Hwang, K.B., Lee, I.H., Kim, J.I., & Kong, S.W. (2015). I148M variant in PNPLA3 reduces central adiposity and metabolic disease risks while increasing nonalcoholic fatty liver disease. *Liver International, 35*(12), 2537–2546. http://doi.org/10.1111/liv.12909

Petta, S., Miele, L., Bugianesi, E., Cammà, C., Rosso, C., Boccia, S., Cabibi, et al. (2014). Glucokinase Regulatory Protein Gene Polymorphism Affects Liver Fibrosis in Non-Alcoholic Fatty Liver Disease. *PLOS ONE, 9*(2), e87523. http://doi.org/10.1371/journal.pone.0087523

Rafeey, M., Mortazavi, F., Mogaddasi, N., Ghaffari, S., & Hasani, A. (2009). Fatty liver in children. *Therapeutics and Clinical Risk Management*, 371–374. http://doi.org/10.2147/TCRM.S4467

Roberts, E. A. (2007). Pediatric nonalcoholic fatty liver disease (NAFLD): A "growing" problem? *Journal of Hepatology, 46*(6), 1133–1142. http://doi.org/10.1016/j.jhep.2007.03.003

Santoro, N., Kursawe, R., D'Adamo, E., Dykas, D. J., Zhang, C. K., Bale, A. E., Cali, A., et al. (2010). A common variant in the patatin-like phospholipase 3 gene (PNPLA3) is associated with fatty liver disease in obese children and adolescents. *Hepatology, 52*(4), 1281–1290. http://doi.org/10.1002/hep.23832

Sarkhy, A., Nobili, V., & Al-Hussaini, A. (2014). Does vitamin E improve the outcomes of pediatric nonalcoholic fatty liver disease? A systematic review and meta-analysis. *Saudi Journal of Gastroenterology, 20(*3), 143–153. http://doi.org/10.4103/1319-3767.132983

Schwimmer, J. B., Pardee, P. E., Lavine, J. E., Blumkin, A. K., & Cook, S. (2008). Cardiovascular Risk Factors and the Metabolic Syndrome in Pediatric Nonalcoholic Fatty Liver Disease. *Circulation, 118*(3), 277–283. http://doi.org/10.1161/CIRCULATIONAHA.107.739920

Schwimmer, J. B., Celedon, M. A., Lavine, J. E., Salem, R., Campbell, N., Schork, N. J., Shiehmorteza, M. (2009). Heritability of Nonalcoholic Fatty Liver Disease. *Gastroenterology, 136*(5), 1585–1592. http://doi.org/10.1053/j.gastro.2009.01.050

St-Jules, D. E., Watters, C. A., Nagamori, K., & King, J. (2013). The Effect of Weight Loss on Pediatric Nonalcoholic Fatty Liver Disease, The Effect of Weight Loss on Pediatric Nonalcoholic Fatty Liver Disease. *International Scholarly Research Notices*, 2013, e398297. http://doi.org/10.1155/2013/398297, 10.1155/2013/398297

Takahashi, Y. (2010). Pediatric nonalcoholic fatty liver disease: Overview with emphasis on histology. *World Journal of Gastroenterology, 16*(42), 5280–5285. http://doi.org/10.3748/wjg.v16.i42.5280

Tan, H.-L., Zain, S. M., Mohamed, R., Rampal, S., Chin, K.-F., Basu, R. C., Cheah, P.L., et al. (2013). Association of glucokinase regulatory gene polymorphisms with risk and severity of non-alcoholic fatty liver disease: an interaction study with adiponutrin gene. *Journal of Gastroenterology, 49*(6), 1056–1064. http://doi.org/10.1007/s00535-013-0850-x

Utzschneider, K. M., & Kahn, S. E. (2006). The Role of Insulin Resistance in Nonalcoholic Fatty Liver Disease. *The Journal of Clinical Endocrinology & Metabolism, 91*(12), 4753–4761. http://doi.org/10.1210/jc.2006-0587

Valenti, L., Alisi, A., Galmozzi, E., Bartuli, A., Del Menico, B., Alterio, A., Dongiovanni, P., et al. (2010). I148M patatin-like phospholipase domain-containing 3 gene variant and severity of pediatric nonalcoholic fatty liver disease. *Hepatology, 52*(4), 1274–1280. http://doi.org/10.1002/hep.23823

Veena, J., Muragundla, A., Sidgiddi, S., & Subramaniam, S. (2014). Non-alcoholic fatty liver disease: need for a balanced nutritional source. *British Journal of Nutrition, 112*(11), 1858–1872. http://doi.org/10.1017/S0007114514002591

Younossi, Z. M., Stepanova, M., Afendy, M., Fang, Y., Younossi, Y., Mir, H., & Srishord, M. (2011). Changes in the Prevalence of the Most Common Causes of Chronic Liver Diseases in the United States From 1988 to 2008. *Clinical Gastroenterology and Hepatology, 9*(6), 524–530.e1. http://doi.org/10.1016/j.cgh.2011.03.020

Zhan, Y.-T. (2010). Roles of liver innate immune cells in nonalcoholic fatty liver disease. *World Journal of Gastroenterology, 16*(37), 4652–4660. http://doi.org/10.3748/wjg.v16.i37.4652

DETERMINISTIC CHAOS

APPLICATIONS IN CARDIAC ELECTROPHYSIOLOGY

BY MISHA KLASSEN

I. INTRODUCTION

Our universe is a complex system. It is made up of many moving parts as a dynamic, multifaceted machine that works in perfect harmony to create the natural world that allows us life. The modeling of dynamical systems is the key to understanding the complex workings of our universe. One such complexity is chaos: a condition exhibited by an irregular or aperiodic nonlinear deterministic system. Data that is generated by a chaotic mechanism will appear scattered and random, yet can be defined by a system of nonlinear equations. These mathematical equations are characterized by their sensitivity to input values (initial conditions), so that small differences in the starting value will lead to large differences in the outcome. With deterministic chaos, it is nearly impossible to make long-term predictions of results.

A system must have at least three dimensions, and nonlinear characteristics, in order to generate deterministic chaos. When *nonlinearity* is introduced as a term in a deterministic model, chaos becomes possible. These nonlinear dynamical systems are seen in many aspects of nature and human physiology. This paper will discuss how the distribution of blood throughout the human body, including factors affecting the heart and blood vessels, demonstrate chaotic behavior.

The physiological studies presented in this paper represent some of the investigations into the chaotic systems that can be found in the human body. With modern computing technologies, we are able to identify patterns that were previously thought to be random variations of regular systems, such as the heartbeat. By understanding these systems on a

mathematical level, scientists can produce mathematical models of irregular oscillations within the body. Currently, research is being conducted to develop chaos control techniques to treat patients with heart rhythm irregularities. This paper will first introduce chaos theory in a historical context, and then present some of its modern scientific applications.

II. CHAOS THEORY

In the 1880s, Henri Poincare was studying the motion of an asteroid under gravitational pull from Jupiter and the sun. The most effective way to investigate the behavior of such a system was to use nonlinear differential equations [16]. These equations were first developed by Sir Isaac Newton in the 1600s, but were heavily studied throughout the 1700s and 1800s [20]. Poincare recognized that in order to model a physical system that evolves over time, one must use a sufficient list of parameters to be able to define the state of the system at any given moment. The values of data measured in time can be made into an object in space, called *the phase space set*. In this case, the phase space set is the set of all possible positions and velocities of the asteroid. Poincare's model was known as the "return map" of the asteroid. This marked an important moment in the timeline of mathematical history by recognizing the sensitivity to initial conditions that models, such as those of the solar system, necessarily demonstrate [1]. Mathematicians studied Poincare's return map, and found that small differences in the initial conditions will lead to very large differences in final outcomes. Therefore, predictions of an asteroid's location based on estimations of its initial conditions are impossible [1]. This was the first time the existence of chaos in natural phenomena was formally recognized by the scientific community.

In the 1920s, Dutch physicist Balthasar Van der Pol modeled an oscillator with nonlinear damping by constructing electrical circuits according to the differential equation [16]:

Dynamical System
A mathematical model used to determine the state of a system as it moves forward in time.

Nonlinear Damping
The damping term of a differential equation is multiplied by the first derivative, causing the motion of the system to decay over time. Nonlinear damping implies that this term is not linear, so the effect of this damping fluctuates over time.

Oscillator
A generator of periodic motion or electrical currents

$$\frac{d^2x}{dt} - \epsilon\,(1-x^2)\,\frac{dx}{dt} + x = 0$$

In this model, t is time, x is the dynamic variable, and ϵ is the parameter that can increase or decrease the influence of the nonlinear term. This can be converted to a first order system by letting $y = \frac{dx}{dt}$

$$\frac{dx}{dt} = y$$

$$\frac{dy}{dt} = -x + \epsilon(\,1 - x^2\,)y$$

The parameter ϵ allows for increased or decreased levels of non-

linearity, dependent on the system being modeled. Leonhard Euler first proposed this method of solving second order equations by reducing them to first order in the 1700s, when he developed the technique of using an integrating factor to solve differential equations [20]. Van der Pol also examined the response of the oscillator to periodic forcing, modeled as:

$$\frac{d^2x}{dt^2} - \epsilon\,(1-x^2)\,\frac{dx}{dt} + x = F\cos\frac{2\pi t}{T_{in}}$$

where ϵ determines the frequency of self-oscillations, while the $F\cos\frac{2\pi t}{Tin}$ term introduces a frequency of periodic forcing [15]. *Tin* is the term for an induced electrical current. With this system, Van der Pol found irregularities in the electrical impulses that he could hear by inserting telephone receivers into the circuits [15]. When periodic forcing is added to a system, its solutions behave seemingly unpredictably [16]. This had been seen already with Poincare's return map of the asteroid, where the periodically varying gravitational forces on the asteroid demonstrated a similar effect. Although Van der Pol did not identify the underlying structure of a chaotic system, the irregularities in his circuit were an example of deterministic chaos, demonstrated in this system when nonlinearity is sufficiently strong [15].

In the 1960s, Edward Lorenz was studying meteorology at the Massachusetts Institute of Technology (MIT). Somewhat accidentally, Lorenz came across the phenomenon of sensitivity to initial conditions; he noted that the same calculation, when rounded to three-digit rather than six-digit figures, came to different solutions that were amplified exponentially with iterative multiplications [1].

In his study of the initial conditions and subsequent modeling of weather patterns, Lorenz saw the same results with these climate models that he had seen in the dynamics of his own calculations. Lorenz presented his models of chaotic systems in 1972, when he introduced the concept of the butterfly effect. This concept is one of sensitivity to initial conditions: the mere flap of a butterfly's wings may drastically affect global climate systems. The computer graphic of his chaotic system was the first representation of an "attractor" [1], which is a specific set of values toward which a system evolves. It was here, thanks to Lorenz, that chaos theory was born. A *Lorenz attractor* is a common model now used to represent chaotic systems similar to those of climate dynamics.

Since the 1960s and the advancement of computing technology, we have been able to create models for many systems in a similar way to Edward Lorenz's foundation of the chaotic attractor. Though much climate and biological data appear to have been generated randomly by the universe, it can actually be modeled with nonlinear dynamical systems, and often generates chaos.

Though random data sets and those that are generated by chaotic mechanisms may look very similar, there are ways to tell whether or not a system demonstrates deterministic chaos. In order to differentiate between random and chaotic data sets, one must first look at the phase space set of each system. Let each point in a phase space have coordinates $x = z(n)$ and $y = z(n+1)$. If the phase space set fills the two-dimensional space with scattered points, this would indicate that the set of data was

generated by a random mechanism. Its fractal dimension will be very high, meaning that the number of independent variables needed to determine a relationship for the data is infinite. If the set does not fill up the two-dimensional space, it will form some object (an attractor) in space with a low fractal dimension [8]. This would demonstrate a deterministic relationship between n and n+1. The fractal dimension of a phase space set is usually a non-integer value. The smallest integer that is greater than or equal to this value is the number of independent variables in the deterministic relationship [8].

To summarize, in a randomly generated data set there will be an infinite number of factors that determine the results, and therefore the dimension of the phase space set is infinite. In a data set that shows deterministic chaos, the fractal dimension will approach a non-integer constant [8]. This is the essence of chaos theory. As we will see, this theory is applicable to irregularities in the cardiovascular system, and can help us mathematically understand abnormalities of the human heart.

III. CHAOS IN THE CARDIOVASCULAR SYSTEM

The human heart has approximately two billion muscle cells [6]. The sinoatrial node is a group of cells in the right atrium that sends out regular electrical impulses every time the heart beats. These impulses cause the right atrium to contract, and they set a person's heart rhythm, referred to as their regular sinus rhythm. The atrioventricular node is located between the right atrium and right ventricle. This group of cells sends out a secondary set of electrical impulses, causing the ventricles to contract, but leaving enough time (about one tenth of a second) after each beat for the ventricles to fill with blood. These impulses initiate the necessary pulsing of blood throughout the body. It is a delicate system; if the electrical impulses are even slightly off, the systole and diastole (contraction and relaxation) of the heart muscle will be seriously affected. Our bodies must maintain a certain blood pressure to push essential nutrients throughout the bloodstream. Similarly, the hemoglobin in our blood, which carries oxygen to our cells, provides for the vital functioning of our organs. Maintaining healthy blood pressure and heart rate is the essence of our well-being, which is why it is so important for scientists and medical professionals to recognize and resolve errors in the system.

Instances of chaos have been found in many cardiovascular system irregularities, including premature ventricular contractions, atrial fibrillation, bradycardia, tachycardia, and cardiac arrhythmia [11]. Premature ventricular contractions are heartbeats that begin in ventricles and disrupt normal rhythm, causing irregular or skipped beats. Atrial fibrillation is caused by the presence of too many electrical impulses in different locations within the cardiac tissue [12]. The spontaneous contraction of cardiac muscle fibers results in a lack of synchronism between the heartbeat and the pulse, so that there is not enough blood being pumped to the body's cells [12]. Ventricular tachycardia is an

Iterative Multiplications
Calculations that multiply repeatedly, using the previous output as the input for the next multiplication.

Periodic Forcing
A term that represents an external influence on the system that repeats after a defined time interval.

Isotropic Upon Rescaling
The shape of the figure remains the same despite zooming in to a small piece or zooming out to the whole object.

A PERFECT EXAMPLE OF THIS IS THE SNOWFLAKE... IT CONTAINS A SEEMINGLY INFINITE NUMBER OF CRYSTALINE PATTERNS

abnormally rapid heartbeat originating in the ventricles, while bradycardia is an abnormally slow heartbeat. Arrhythmia is a generally abnormal heart rhythm [12]. In this paper, these factors of irregularity are generally referred to as *heart rate variability*.

It is incredible to consider how the minor imperfections and complexities in all living things can in fact be defined by mathematical principles. Most objects we observe in our daily lives have continuous curves, but these curves are typically not differentiable. Self-similarity, the notion of the small patterns reflecting the larger patterns of a system, is demonstrated in abundance in the natural world. A perfect example of this is the snowflake: upon magnification, it contains a seemingly infinite number of crystalline patterns that reflect the image of the flake as a whole, so that it is isotropic upon rescaling [7]. Though we are familiar with Euclidean geometric figures, we often fail to recognize the inherent complexity of the natural structures that surround us. Polish mathematician Benoit Mandelbrot first introduced the term *"fractal"* in 1975 to characterize these forms [7].

Like chaos, a fractal pattern cannot be effectively conveyed with estimation [7]. For example, if maps had curves that estimated the fractal dimension of a shoreline, we would not be able to accurately measure its distance or determine the shape when trying to land a ship. If the inherent complexity of the shoreline is not displayed, the necessary information is lost. Many aspects of our physiology demonstrate fractal patterns. As in the crystals of a snowflake or branches of a willow, these patterns make up our very cells. Self-similarity can be witnessed in the dendrites of nerve pathways, the blood vessels in the retina, and the bronchioles in the lungs. These self-similar patterns throughout the human body may be determined by some very basic rules in our genes. Perhaps this is how our bodies can display millions of such seemingly intricate structures with only 100,000 genes to guide their production [8]. There is evidence that the fractal branching design generates the most efficient way for blood to travel throughout the body by minimizing the work of transport between cells [5]. While the organization of the passage of fluid and electrical signals through the body can be revealed by fractal analysis, the underlying dynamics of the system can be understood by chaos theory.

In his article "Deterministic Chaos and Fractal Complexity in the Dynamics of Cardiovascular Behavior," Vijay Sharma writes, "Deterministic chaos describes a system which is no longer confined to repeating a particular rhythm, and is free to respond and adapt" [5]. Our heart is a perfect example of this phenomenon. According to Sharma, the interaction of calcium oscillators in the cytoplasm initiate rhythmic changes in the diameter of blood vessels, which exhibit chaotic behavior. Decreasing the local pressure within vessels by increasing their diameter will induce regular periodic dynamics (i.e. non-chaotic behavior). When resistance vessels are activated, chaotic

behavior will initiate. Since the sympathetic nervous system regulates the level of the resistance vessels, its activity is one of the contributing factors to chaotic behavior in vasomotion [5]. Using techniques that control initiation of the sympathetic nervous system, this chaotic behavior can be artificially induced in a lab. It has also been shown that chaotic behavior increases with increased activity in the parasympathetic nervous system. This may be due to the fact that short-term variation in heart rate is predominantly influenced by the parasympathetic nervous system. Therefore, both sympathetic and parasympathetic nerve pathways affect long-term heart rate variability, and the variability can be modeled using deterministic systems [11].

With perfusion pressure control mechanisms, the chaotic behavior can be physically increased or decreased. Various drugs that affect blood vessels can change the heart rate's sensitivity to initial conditions. Studies of mammalian blood pressure variability show that nitric oxide inhibitors, for example, decrease the chaotic behavior of the system. In 2012, Siroos Nazari and his colleagues at Payame Noor University in Tehran modeled the intrinsic pacemakers in the heart muscle: the sinoatrial (SA) node, atrioventricular (AV) node, and the His-Purkinje bundles. The SA node is recognized as the primary natural pacemaker of the heart; it is located in the right atrium and generates an electrical current of 60-100 beats per minute. The AV node and His-Purkinje bundle function as a secondary system between the right atrium and right ventricle and beat at a slower rate [12].

Nazari claims that the dynamic behavior of the heart is similar to the Van der Pol oscillator. The model they present uses the Van der Pol equations as a starting point, including a term for periodic forcing that accounts for electrical stimulation of the heart. For the purpose of this paper, I will present the models for the SA and AV nodes as follows and exclude the fifth and sixth equations for the His-Purkinje bundle [10].

Sensitivity to Initial Conditions
A characteristic of a system that causes minor changes in the input values to yield drastically different results. This can be tricky, since a small error in numerical approximation to initial conditions can demonstrate drastically different long-term system behavior.

Periodic Forcing
A term that represents an external influence on the system that repeats after a defined time interval.

Vasomotion:
The oscillating changes in the diameter of blood vessels. Vasomotion affects blood pressure independently from the hearbeat.

Sinoatrial Node:

$$\frac{dx_1}{dt} = y_2$$

$$\frac{dy_1}{dt} = -d_1(x_1^2-1)y_1 - c_1x_1 + a_1\cos wt + R_1(x_1-x_3)$$

Atrioventricular Node:

$$\frac{dx_3}{dt} = y_2$$

$$\frac{dy_2}{dt} = -d_2(x_3^2-1)y_2 - c_2x_3 + a_2\cos wt\, R_2(y_1-y_2)$$

The parameter w represents external electrical stimulation and gives the term for oscillator frequency. The coefficients d_1 and d_2 affect the nonlinearity of the system and cause stability of the limit cycle. A more stable limit cycle results in values that return quickly to the attractor. c_1 and c_2 represent the frequencies of the SA node and AV node, respectively. R_1 and R_2 are the coupling coefficients between nodes. An example of this relationship would be if $R_1 = 0$ and $R_2 > 0$, then the SA node has an effect on the AV node, but not vice versa. In another case, if $R_1 > R_2$, then the AV node has a greater effect on the SA node [10].

Numerical simulations were carried out in a computer program to demonstrate that the model they developed does capture the dynamics of regular sinus rhythm. Various heart conditions were taken into account by manipulating the coupling coefficients to represent different interactions of the SA and AV nodes. Nazari and his fellow scientists concluded that their heart model could be chaotic or non-chaotic, depending on the size of the nonlinearity parameters, d_1 and d_2 [10].

Twenty years prior to Nazari's work, research concerning heart rate variability had sparked the interest of the mathematical community in North America. In 1990, Leon Glass and his colleagues at McGill University studied heart cell aggregates in chicks and the overdrive suppression evident after periodic electrical stimulation of the heartbeat. Overdrive suppression occurs when the heart rate is overstimulated to the point that the intrinsic frequency of spontaneous heart rhythm actually slows. This occurs naturally in the heart, where the spontaneous electrical activity of the SA node is of a higher frequency than the activity in other nodal firing sites, such as the AV node. The rapid firing of the SA node creates an increased level of sodium ions, resulting in a chain of chemical events that prevent the spontaneous generation of beats in the other pacemaker sites [18]. Various other ionic mechanisms play a role in overdrive suppression, including extracellular and intracellular calcium and potassium imbalance [13]. This study investigated the effects of artificial stimulation frequency, amplitude, and variation. The scientists measured the number of beats between electrical stimulations, and found that the spontaneous beats varied with the frequency of artificial stimulations. Both artificial impulses and spontaneous beats were found to be coupled in the integer ratios of 1:1, 2:1, and 2:3, depending on the frequency of artificial stimulations, demonstrating a nonlinear response [8].

The mathematicians involved in this research came up with a system of nonlinear differential equations to model their results. They began with a piecewise linear approximation to the Van der Pol equations to represent the cardiac cycle. Other biological oscillators have been modeled in this way since Van der Pol's work with simple sets of ordinary differential equations [13]. This model is written as

$$\frac{dV}{dt} = \frac{1}{\epsilon}[y - f(V)]$$

$$\frac{dy}{dt} = a(V)$$

where $V(t)$ is the experimentally observed transmembrane voltage. This is the measurement of electrical activity in the heart. Essentially, $V(t)$ is the calculation of movement of positive ions from intracellular to extracellular space. This change in voltage

is referred to as an action potential. $f(V)$ and $a(V)$ are piecewise linear functions of V. ϵ is a positive constant parameter. When ϵ is small ($0 < \epsilon << 1$), the oscillations will quickly return to the attractor [13].

Since the main assumption of this study is that overdrive suppression is a consequence of the outward electrical current, the researchers had to include a term for a history-dependent hyperpolarizing current (Z). After initiation of an action potential, the heart muscle undergoes a refractory period so that the ventricles can refill completely with blood. This is the depolarization of the heart cell membranes; they close and ionic movement becomes inactive. However, if a cell is hyperpolarized, the membrane threshold potential will become more negative and it will take a stronger than normal stimulus for the cell membrane to open and for an action potential to occur. Increasing the electrical stimulus that induces a heartbeat will increase the number of hyperpolarizing currents generated [18]. The Z term takes into account any previous hyperpolarization of the heart cells. So, with each induced stimulus, spontaneous generation of action potential is inhibited and the cardiac refractory period will be lengthened.

A term is now added to the second ordinary differential equation (the term $\beta \frac{Z}{Z+k}$) that affects y in the second equation, and results in a longer duration of the depolarizing phase of the cardiac cycle. This is where overdrive suppression is introduced into the model. In addition, the parameters β and y are introduced as positive constants, and ΔZ is an instantaneous positive increment that comes from the onset of action potential (at time t_{AP}) during ionic movement. δ is the Dirac delta function [21].

The final model presented in this study is as follows [13]:

$$\frac{dV}{dt} = \frac{1}{\epsilon}[y - f(V)]$$

$$\frac{dy}{dt} = a(V) - \beta \frac{Z}{Z+k}$$

$$\frac{dZ}{dt} = -y \frac{Z}{Z+k} + \Delta Z \delta (t - t_{AP})$$

The experiment resulted in the understanding that oscillators within the cardiovascular system demonstrate minor variability that is very sensitive to initial stimulus. The time of stimulus has a similar effect on these oscillators, as does periodic forcing on the Van der Pol oscillator. There is evidence that this

Euclidean Geometry
First introduced in Euclid's famous book *The Elements* (~300 B.C). We are familiar with the constructing of lines, circles, and regular polygons, but these figures are uncommon in the natural world. Non-Euclidean geometry includes the fractal patterns discussed in this paper.

Piecewise Function
A function made up of smaller functions on sequential intervals that make up the domain of the function as a whole.

complex evolution of the rhythmic pattern may apply to other oscillating biological systems under periodic stimulation [19].

IV. CONCLUSION

Throughout our world, the intrinsic value of natural biological processes can be seen through chaos theory. From the fractal patterns in the naked branches of trees to the orbiting asteroids in outer space, deterministic chaos plays a role in the character of life. With this century's modern computing methods, we are able to capture and model these systems in a way that scientists never could before, allowing for medical innovations that may change how humans respond to physiological concerns.

Original methods used artificial pacemakers that induced large electrical currents to force the heart out of irregularities [8]. We can, however, use smaller electrical currents that are applied at specific intervals computed from the deterministic relationship between these stimulations and heartbeats. Chaos control techniques can now be employed by scientists to fix the medical complications caused by abnormal heart rate variability. Smaller impulses in pacemakers can be used to stabilize the heartbeat; the technique of subtle perturbations can be used to stabilize any biological oscillator that demonstrates chaotic behavior [11]. By explicitly modeling the impulses of the human heart, we are able to apply chaos control techniques to modern pacemakers, and attempt to reduce the risk of heart attacks and other potentially life-threatening cardiovascular problems.

REFERENCES

[1] Oestreicher C. (2007). *A history of chaos theory*. Dialogues Clin Neurosci.

[2] Williams, G.P. (1997). "*Chaos Theory Tamed*." Washington D.C.: Joseph Henry Press.

[3] Goldberger A. L., Amaral L. N., Glass L., Hausdorff J.M., Ivanov P.C., Mark R.G., Mietus J.E., Moody G.B., Peng C-K., Stanley H.E. (2000, June 3). *Components of a New Research Resource for Complex Physiologic Signals*. Circulation, 101(23), e215-e220. Retrieved from http://circ.ahajournals.org/cgi/content/full/101/23/e215

[4] Elbert T., Ray WJ, Kowalik ZJ, Skinner JE, Graf KE, Birbaumer N. *Chaos and physiology: deterministic chaos in excitable cell assemblies.* Institute for Experimental Audiology, University of Münster, Germany 1994.

[5] Sharma, V (2009). *Deterministic chaos and fractal complexity in the dynamics of cardiovascular behavior: perspectives on a new frontier*. Division of Pharmacology and Toxicology, Faculty of Pharmaceutical Sciences, The University of British Columbia.

[6] Adler, C.P., Costabel, U. (1975). *Cell number in human heart in atrophy, hypertrophy, and under the influence of cytostatics*; 6:343-55. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/128080

[7] Kenkel, N.C. (1996). Walker, D.J. *Fractals in the Biological Sciences*. Retrieved from http://www.umanitoba.ca/science/biological_sciences/botany_lab/pubs/1996.pdf

[8] Liebovitch L. (1998). *Fractals And Chaos Simplified For The Life Sciences*. New York: Oxford University Press.

[9] Goldberger, Ary L. et al. (2002). *Fractal Dynamics in Physiology: Alterations with Disease and Aging. Proceedings of the National Academy of Sciences of the United States of America*, 99(1), 2466–2472. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC128562/

[10] Nazari S., Heydari A., Khaligh J. (2013 July). *Modified Modeling of the Heart by Applying Nonlinear Oscillators and Designing Proper Control Signal.* Applied Mathematics, 4, 972-978. Retrieved from http://www.scirp.org/journal/am

[11] Savi, M. A. (2005 Apr-June). *Chaos and Order in Biomedical Rhythms.* Journal of the Brazilian Society of Mechanical Science and Engineering. Vol. XXVII, No. 2, 157-169.

[12] Layton, L. (2008, Sept. 23). *What Determines the Rhythm of Your Heart? How Stuff Works*. Retrieved from http://health.howstuffworks.com/human-body/systems/circulatory/heart-rhythm.htm

[13] Kunysz, A., Glass L.,Shrier A. (1995). *Over-drive suppression of spontaneously beating chick heart cell aggregates: experiment and theory.* Am. J. Physiol. 269 (Heart Circ. Physiol. 38): H1153-H1164.

[14] Nsom, C. T. *The Electrical Activity of the Heart and the Van der Pol Equation as its Mathematical Model.*

[15] Kanamaru, T. (2007) *Van der Pol oscillator*. Scholarpedia, 2(1): 2202. Retrieved from http://www.scholarpedia.org/article/Van_der_Pol_oscillator#Periodic_Forcing_and_Deterministic_Chaos

[16] Blanchard, P., Devaney, R. L., Glen, R. H. (2006). *Differential Equations*, Third Edition. Belmont, CA: Brooks/Cole.

[17] Nikolic, B. K. *Introduction to Deterministic Chaos*. University of Delaware. Retrieved from http://www.physics.udel.edu/~bnikolic/teaching/phys660/lectures/deterministic_chaos.pdf

[18] Klabunde, R.E. (2007, Apr. 6). "*Overdrive Suppression*." Cardiovascular Physiology Concepts. Retrieved from http://www.cvphysiology.com/Arrhythmias/A018.htm

[19] Glass, L., Peter, H., McCulloch, A. (1991). *Theory of Heart*. New York: Springer-Verlag Inc.

[20] Sasser, J.E. n.d. *History of Ordinary Differential Equations: The first hundred years.* University of Cincinnati.

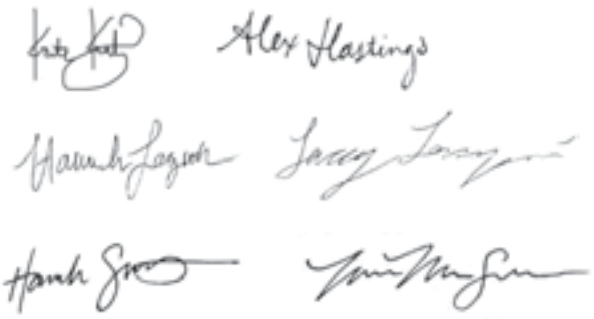
[21] Tornberg, A.K. *The Dirac Delta Function.* Kungliga Tekniska hogskolan. Web. 9 March 2016. http://www.nada.kth.se/~annak/diracdelta.pdf

AFTERWORD

We received a great deal of exceptional submissions for this volume of *Occam's Razor*. The amount of enthusiasm for our magazine was humbling, and spoke to the quality of work that Western students regularly produce. Naturally, this made for a challenging decision-making process. We are inspired by the amount of talent here on campus, and the experience of reading so many diverse submissions was as gratifying as it was enlightening. We hope that you found *Occam's Razor* to be as enriching an experience as we did. We welcome your participation in future volumes; please find our contact and submission information on the inside back cover.

Sincerely,

The *OR* Staff



HOW TO SUBMIT

Interested in submitting to next year's volume of Occam's Razor*? Please visit our website for submission guidelines and deadlines:*
http://wp.wwu.edu/occamsrazor

Read this and past volumes online:
https://issuu.com/occamsrazor

Make sure to "like" us on Facebook:
Occam's Razor WWU

CONTACT US

occamsrazor@wwu.edu
Office: HU 254
Hours on website

VOLUME SIX

Western Washington University 2016