Wandering womb: a real story of endometriosis

Megan Marie Griffiths
Iowa State University

Follow this and additional works at: https://lib.dr.iastate.edu/rtd
Part of the Creative Writing Commons, and the English Language and Literature Commons

Recommended Citation
https://lib.dr.iastate.edu/rtd/16111

This Thesis is brought to you for free and open access by the Iowa State University Capstones, Theses and Dissertations at Iowa State University Digital Repository. It has been accepted for inclusion in Retrospective Theses and Dissertations by an authorized administrator of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
Wandering womb: a real story of endometriosis

by

Megan Marie Griffiths

A thesis submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of
MASTER OF ARTS

Major: English (Creative Writing)

Program of Study Committee:
Mary Swander, Major Professor
Eugenia Farrar
Amy Slagell

Iowa State University
Ames, Iowa
2004
Graduate College
Iowa State University

This is to certify that the master’s thesis of

Megan Marie Griffiths

has met the thesis requirements of Iowa State University

Signatures have been redacted for privacy
## TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Book Proposal</td>
<td>1</td>
</tr>
<tr>
<td><em>Meter: Origins</em></td>
<td>9</td>
</tr>
<tr>
<td>Saltines, Anaprox, and Blackberry Wine</td>
<td>21</td>
</tr>
<tr>
<td>Auto-Destruct</td>
<td>32</td>
</tr>
<tr>
<td>Cavity Search</td>
<td>38</td>
</tr>
<tr>
<td>Demeter</td>
<td>45</td>
</tr>
<tr>
<td>Period</td>
<td>50</td>
</tr>
<tr>
<td>Notes on Sources</td>
<td>59</td>
</tr>
</tbody>
</table>
It hurts. It begins as a tightness, a feeling of pulling in my abdomen, and then rapidly becomes more intense, sharp pulses of painful cramps. I grit my teeth, shift my position in this uncomfortable airport seat, and try to breathe deeply. At the moment, there’s nothing else I can do. I’ve already taken my twice-daily dose of anti-inflammatory drugs, and I sip hot coffee, feeling its warmth travel into my gut. If I were anywhere else, I’d be swathed in two layers of heat wraps—one for my back and one for my front—but I spent some time this morning considering the havoc that would inevitably be wreaked if I walked through the metal detector wearing bands of small iron pellets. I decided to forgo the heat wraps.

So I sip, and breathe, and sip again. I watch people move past, some running, some strolling, all anonymous faces in this ultimate place of anonymity. I sit here, alone, my face carefully set into a mask of painlessness, and I wonder how many other women in this airport are living with pain, invisible and unacknowledged but real, nonetheless.

The accepted truth in our society, culturally enforced and medically indoctrinated, has been that this monthly pain, for women, is normal. When a girl first begins her period, she is told that she is now a woman, and that with this passage come pain and discomfort. Her mother, perhaps, or her sister or aunt, teaches her about hot tea, heating pads, and Midol. This girl—young and frightened by an event over which she has no control—resigns herself to this fate. Perhaps she has pain. Perhaps she curls up in bed, her muscles clenched in tension, her breath hitching with each cramp. Perhaps she cries. Quite possibly, she will tell her mother, who may reassure her that this pain is normal. Or perhaps her mother, concerned for her daughter, will accompany her to the doctor, who will smile at them both, babble about
what’s involved in being a woman, and then dole out birth control pills. This girl and her mother may accept this. Or they may fight back.

This book is for those who fight back, or who long to.

I have lived with this monthly pain since I first began menstruating at the age of twelve. It was a full nine years later that the official diagnosis confirmed my own unofficial one: I had endometriosis. This was not unexpected, as my mother, my aunt, and my sister also had the disease. The diagnosis, however, also placed me within a larger community of women connected by this odd “wandering womb” disease—a disease in which the lining of the uterus grows in places where it shouldn’t, causing such problems as severe monthly pain, pain during sex, infertility, and a host of immune-related diseases.

Endometriosis is a chronic disease found in at least ten percent of women worldwide—perhaps as many as 89 million women—making it more common than AIDS and cancer. While it was recognized as a disease as early as 1690, for years women have been battling doctors who insisted that there was nothing wrong, that monthly pain was normal, or that childbirth would solve the problem. Although endometriosis is finally becoming better understood, research still has not yielded a cure or even a reliable treatment.

Because of this lack of support from the medical community, women with the disease have turned to each other—thanks largely to the Internet—for advice and compassion. The Endometriosis Association (EA), an international organization, has published books about the disease to educate women about the complexities of diagnosis and treatment. Other groups, such as the Endometriosis Research Center (ERC), keep women updated on advances in research through e-mail and message boards.
As a woman with endometriosis and a student of biology, I am both repelled by and fascinated with endometriosis. The disease is wily and tenacious, and resists definition. But as I began my research into the disease, my fascination grew to include the women who have this condition. Many of them, though suffering with intense pain, regularly comfort others, lobby Congress for more funding, contribute money and time to endometriosis support groups, and educate people about this misunderstood disease.

It is for them—and that frightened twelve-year-old girl—that I write this book. There are few books that discuss endometriosis both scientifically and personally, and this is what I wish to do. I hope that women already well-versed in the science of endometriosis will find the book to be a clear, strong summary of the disease's history and pathology; I also hope that women who know little about the disease will use the book as a guide and a comfort. The book could be lugged to doctors' appointments, but then replaced on the nightstand for bedtime reading.

*Wandering Womb* will examine the proposed causes of endometriosis, how it has been perceived and treated historically, problems with diagnosis, and its connection to infertility. Because recent research suggests that endometriosis is most likely an inherited disease, the book will also explore the effects of the disease in the lives of my mother, my aunt, and my sister, and will examine studies specifically addressing the role of genetics in endometriosis. The text will also address the emerging evidence suggesting that endometriosis is closely tied to the immune system.

The book, then, will chronicle my own experience with endometriosis in the larger context of the history, pathology, and etiology of the disease. It will be a memoir, but will also feature a significant amount of scientific information, written for the layperson. Its
ultimate message will be one of admiration for both the disease and the women who have it, and of hope for an eventual cure.

**Structure**

The book's chapters will each tackle one aspect of endometriosis, and in these chapters, my own story will also be told throughout the book. I will begin, then, with the ongoing debate over what causes endometriosis (its origins), moving next to the variety of treatments for endometriosis. I will then examine recent research into a possible link between endometriosis and autoimmune disease. Some time will be spent discussing the use of laparoscopy and hysterectomy to both diagnose and treat endometriosis. Finally, I will look more closely at endometriosis' possible effects on fertility and where the research on the disease stands now. Scientific, historical, and sociological information will be the main focus of each chapter, with my own experiences serving to frame the chapters.

**Chapter Sketches**

*Meter: Origins:* The book will begin with a diagnosis—not of my own endometriosis, but of my sister's. Her case is unusual in that she did not experience symptoms before being diagnosed with the worst stage of the disease. This simply illustrates the inexplicable nature of endometriosis: its effects, as well as its origins, are poorly understood, even after years of research. This chapter will examine the various theories regarding the possible causes of endometriosis, as well as the origins of the disease within my own family.
Saltines, Anaprox, and Blackberry Wine: The parade of doctors begins as I attempt to deal with the effects of endometriosis from the age of twelve until the age of twenty-two. I will discuss the possible reasons for the pain associated with endometriosis and then the treatment program that most doctors prescribe: birth control pills, pain relievers, birth control pills, drugs to induce pseudo-menopause, and more birth control pills. In addition, I’ll explore the frustration, common to nearly all women with endometriosis, of finding the medical community unsupportive, insisting that the pain is exaggerated or “all in your head.”

Auto-Destruct: This will be a chapter examining recent research which suggests that endometriosis is part of an underlying auto-immune disorder. Women with endometriosis often find that they are more likely to also develop rheumatoid arthritis, allergies, fibromyalgia, chronic fatigue syndrome, diabetes, and a host of other illnesses. Many researchers believe that unraveling the connection between endometriosis and the immune system will pave the way for an eventual cure.

Cavity Search: The only way to diagnose endometriosis, as of right now, is through a laparoscopy, an outpatient surgical procedure. Additionally, many women resort to a hysterectomy as a final, desperate attempt to finally rid themselves of the disease. In this chapter, I will take readers through the procedure of my own laparoscopy, as well as the mixed emotions following my official diagnosis of endometriosis. I’ll explore the use of laparoscopies and hysterectomies in the diagnosis and treatment of endometriosis, as well as the common myths about these surgeries, and what women can reasonably expect if they choose the surgical route.
Demeter: This chapter will examine the relationship between endometriosis and infertility. Research suggests that about one-third of women with endometriosis experience infertility, while about one-third of women experiencing infertility are found to have endometriosis. Many women don’t even discover they have endometriosis until they are unable to conceive; the diagnosis is a devastating blow. The causes behind endometriosis-related infertility are still unclear, though research is ongoing.

Period: The closing chapter will be one of connections, updates, and hope. I’ll explore what is going on now, both in endometriosis research and in the lives of those affected by endometriosis, including my family members. This chapter will be about strength and resilience, showing what women with endometriosis have accomplished both in the political world and in terms of educating the public. The closing message will be one of encouragement and hope.

Market and Audience

While women’s health books have become a major presence in the nonfiction market in recent years, the majority of these books focus on three things: infertility, pregnancy, and menopause. While the books on infertility often discuss endometriosis as a possible cause for the problem, they rarely discuss the disease in great depth, or in any context other than the disease’s possible detrimental effects on fertility.
There are some books about endometriosis on the bookshelves, but nearly all of these books take an impersonal, detached approach to the disease. They offer an extensive scientific background, practical and sound advice for dealing with endometriosis, and clear descriptions of the plethora of theories surrounding the disease, therefore making them extremely useful references, but they seldom attempt to make the disease seem personal. The books occasionally include letters or testimony from women affected by the disease, which does serve to personalize endometriosis, but these stories are merely snippets of women’s experiences and fail to tell the whole story. Additionally, many of the books focus primarily on one aspect of the disease, such as the psychological effects of endometriosis (*Coping with Endometriosis*) or the possible effects of diet on the disease (*Endometriosis: A Key to Healing through Nutrition*). While these would be useful to a reader seeking only specific information, they won’t be as helpful to someone who is looking for a more extensive explanation of the disease. On the flip side, expansive books like the Endometriosis Association’s *The Endometriosis Sourcebook* and *Endometriosis: The Complete Reference for Taking Charge of Your Health*, while excellent books on the topic, are overwhelming to the casual reader.

One memoir on endometriosis has already been written. It is called *Endometriosis: One Woman’s Journey*, by Jennifer Marie Lewis, and was published in 1998. Since 1998, however, significant advances have taken place in endometriosis research, making a new book not only helpful, but necessary to women seeking the most recent information. Lewis’ book is also limited in that it skims quickly over the possible causes, symptoms, and treatments, raising more questions than it answers. It does not discuss, at all, the possible link between endometriosis and the immune system. Approximately half of the book is
composed of letters women have written to Lewis concerning their own experiences which, while interesting and affirming, do not offer any new information or insights into the disease.

My hope is that women with endometriosis, as well as those who have loved ones with the disease, will read *Wandering Womb* and find it a clear, concise, up-to-date explanation of the various aspects of endometriosis, and that my own story will serve simply to ground the information in one continuous narrative.

**Biography**

Megan Griffiths is a 2001 graduate of Hiram College in Hiram, Ohio. She received her Bachelor’s degree in biology, with a minor in writing. Her biology courses at Hiram were mainly focused on genetics and cellular biology, while her writing courses ran the gamut from nonfiction writing to playwriting. Her interests in endometriosis research and science writing were both sparked in an advanced molecular biology course when she worked on an in-depth analysis of the possible roles of integrins (cell-adhesion molecules) and matrix metalloproteinases (enzymes that initiate extra-cellular matrix breakdown) in the spread of endometrial tissue. Research into this area soon spread to an interest in other aspects of the disease, since endometriosis had been diagnosed in her mother and her aunt. Shortly after she and her sister were also diagnosed with the disease, she began her Master’s work in creative writing at Iowa State University. She anticipates graduating in May 2004.
August 2002

We're waiting. We've been waiting for a long time.

The hospital has taken great pains to transform this waiting area into tiny living rooms, complete with TVs and coffee tables. People lie stretched out on couches, or slouch down into overstuffed chairs, their faces blank. Discarded newspapers and partially completed crossword puzzles have piled up on the floor.

My mother sits on the edge of the couch in our little corner. My father leans back into his chair, his legs crossed and eyes closed. I share the couch with my mother, but I do not sit still.

The enforced living room atmosphere grates on me. There is a layer of tension, a collective held breath each time a patient's family is paged. CNN blares from numerous televisions, but no one is listening. Families who have already been consulted laugh and play cards next to families with drawn faces and restless, tapping fingers.

We do not point out the obvious, my parents or I; we do not comment that my older sister has been in surgery, this simple outpatient procedure, much too long. We all know. I munch through a second bag of Doritos, trying to keep busy, as my mother closes her eyes and my father begins to scan the newspaper again.

It has been three hours by the time we hear "Family of Carrie Griffiths" over the loudspeaker. We walk up to the desk, announce who we are, and are shown into a small, stuffy, but richly furnished consulting room. A moment later, the surgeon enters, still in his scrubs. He nods a greeting. We nod back.
“She has Stage Four,” he says.

My parents look at the doctor blankly, until finally my mother asks, “Stage Four what?”

The doctor seems surprised by the question. “Stage Four endometriosis. Here, these are the pictures—I’ll draw you a diagram of how it looked.”

He pulls out a sheet of paper and, as he sketches, he points to the photos of my sister’s blackened, misshapen ovaries and talks about adhesions and bladders and cysts.

“She had an implant the size of a grapefruit”—he makes a large ‘O’ with his hands—“attached to her right ovary, which is the size of a grape”—he makes an ‘o’ with his first finger and thumb. “And she had adhesions all over her bowel and her other ovary. Definitely Stage Four—lucky we went in there to clean it out.”

Infertility is a possibility, he says; carrying a child to full-term won’t be easy. He points at some more structures on his diagram and continues talking to my parents as I stare at the photographs.

I’m not squeamish; I’d looked at my own laparoscopy photographs two months earlier with fascination. There was the ovary, ready for ovulation with an egg pushing against the outer wall like a child within the womb, there was the uterus, its tissue a healthy pink. There were the tiny black spots of endometriosis. But as I look now at my sister’s photographs, I feel sick. The tissue is dark, her uterus and ovaries obscured by thin black fog. Endometriosis has taken over the photographs, just as it has taken over my sister’s abdomen. I have to turn away from the pictures.

As we leave the small consulting room, stunned, my mom whispers to me, “Do you think he even remembered what the surgery was supposed to be for in the first place?”
I shrug. It’s a fair question, since my sister had only undergone the laparoscopy to have a large cyst removed, a cyst that her gynecologist had detected during a routine physical exam. It was almost certainly not endometriosis, the doctors had agreed, since it’s nearly impossible to feel the signs of endometrial adhesions during a physical examination. Besides, my sister had never shown any symptoms of the disease. The surgeon, who had performed my laparoscopy just two months earlier, was a specialist in endometriosis, but he agreed to take my sister’s case even though it wasn’t endometriosis-related.

But now he’s telling us that it is, that the grapefruit-sized cyst is a huge adhesion and that my sister has the worst stage of the disease.

“I can’t believe Carrie has endometriosis,” my mother says to no one in particular as we return to the waiting room.

It may sound callous, but before that surgery commenced, I would have been willing to put money on the fact that they would find endometriosis in my sister. Disease, like history, often repeats itself. My mother and her sister had both been diagnosed with endometriosis, and now, so had my sister and I. Heredity is inescapable, the bad passed on along with the good.

The Center for Fertility and Reproductive Endocrinology at Virginia Mason Medical Center in Seattle suggests that, if your mother and sisters have endometriosis, you are ten times more likely to have the disease yourself. My sister is one of the luckiest people I’ve ever known. She was once dealt four aces in a hand of five-card draw, but even she couldn’t escape these overwhelming odds.

It’s difficult to say what a woman’s likelihood for having endometriosis is even if she doesn’t have a family history of the disease, because there are no hard-and-fast statistics on
record. Nobody really knows how many women have endometriosis. Some have suggested 5% of women worldwide; others, 20%. Some say the incidence of the disease might be even higher. The most recent Endometriosis Association estimate suggests that at least 89 million women in the world have endometriosis.

So what is it? What is this disease that has interfered in the lives of countless women around the globe?

Simply put, endometriosis is the problem of certain cells gathering where they shouldn’t. The cells that make up the lining of the uterus are called, collectively, the endometrium. In someone with endometriosis, those cells, for some still-unexplained reason, start to grow in places other than the uterus. They form into nodules, and vary in appearance depending on their age and level of activity: some are clear, some red, some brown (called “chocolate” cysts because they’re filled with dried blood), some black (called “powder burns”). The nodules can be microscopic or the size of a grapefruit. They eventually develop blood vessels and can begin to infiltrate surrounding tissue, forming adhesions between themselves, the pelvic organs, and the walls of the pelvic cavity.

Each month, when an egg is released and not fertilized, the body sends out signals telling the uterus that it’s time to shed its lining. The uterus complies, and sloughing begins, sending cells and blood out of the body through menstruation. At the same time, however, those wandering endometrial cells that have taken root on the ovaries, bladder, fallopian tubes, bowel, or other abdominal organs also receive this signal to slough, and they also comply. Unfortunately, when these cells attempt to break down and leave the body, there’s no escape route, so they simply scar over and form implants. For a number of still-unclear reasons, these implants can cause pain, even infertility. And depending on where these
implants are growing, they can also interfere with the normal workings of the bowel, bladder, fallopian tubes, and so on. Oddly enough, the number or size of implants (classified from Stage 1, minimal disease, to Stage IV, severe disease) does not necessarily correlate with the severity of symptoms.

The disease sounds simple, if a bit strange, when explained in this way. If it were this simple, though, endometriosis would not be a medical mystery, poorly understood and under-diagnosed. This simple explanation does not begin to account for the well-documented coupling of endometriosis with other diseases such as allergies, hypothyroidism, rheumatoid arthritis, fibromyalgia, and chronic fatigue syndrome. It offers little insight into why some adhesions cause pain and others don’t. And it fails, on a fundamental level, to explain why, in some women, endometrial cells grow outside of the womb.

So throughout this story, we will have to cut deeper, travel further back into the origins.

Any text you read on the subject of endometriosis is most likely going to start with a discussion of Dr. John Sampson’s theory of retrograde menstruation, proposed back in 1921. Sampson believed that when a woman menstruates, some of that fluid—composed of endometrium and blood—is, to use his word, “regurgitated,” or forced back up, away from the uterus and out into the pelvic cavity, where it is “showered upon the pelvic organs and pelvic lining.” So those endometrial cells travel up instead of down, against the flow, like salmon swimming upstream, and, upon finding themselves in foreign territory, simply drop roots and spawn.

Sampson’s theory was held as fact for a long time—well into the 1970s. Around that time, though, there came further studies that revealed some holes in his hypothesis. For
starters, it was discovered that many—9 out of 10, in fact—women experience retrograde menstruation, yet clearly not all of these women develop endometriosis. Additionally, endometrial adhesions have been found, occasionally, in women’s lungs, lymph nodes, skin, and breasts. There would have to be a mighty powerful burst of backflow to shoot those endometrial cells so far away from the pelvis. A third blow to Sampson’s theory came when researchers discovered that in extremely rare cases, endometriosis was found in the bladders of men who were being treated with estrogens for prostate cancer, as was reported in the *Journal of Urology* in 1971. These men were certainly not victims of retrograde menstruation.

Clearly, then, Sampson’s theory—while helpful in suggesting some cases of endometriosis—was not inclusive enough. Many researchers now believe that it’s the combination of retrograde menstruation along with other factors that leads to endometriosis. If this is the case, the question becomes clear: what are these other factors?

One idea is that endometrial cells are spread throughout the body by traveling through the bloodstream or lymphatic system. This would, of course, explain the presence of the cells in places such as the lungs and lymph nodes. But it does not explain why those cells turned traitor in the first place, or how in the world they can develop in men. This theory goes a long way in illuminating how the disease may spread, but to find out how it actually begins, we’ll have to keep looking.

And so on to the theory of “Coelomic Metaplasia,” proposed by Drs. Ivanoff and Meyer in 1927, only six years after Sampson’s theory. This strange theory suggests that cells, when hit with the right stimulant, can transform themselves into other types of cells. When a fetus is developing, it contains coelomic cells, which are the cells that eventually
turn into either the endometrium or the cells that make up the lining of the abdominal cavity (peritoneal cells, which both men and women have). This metaplasia theory suggests that, once a person has grown, some of those peritoneal cells change into endometrial cells. Why? No one knows. But this theory does offer an explanation for the extremely rare appearance of endometrial cells in men.

Recently, researchers have been gathering evidence that endometriosis may be part of an underlying auto-immune disease, a condition in which the body attacks its own cells, thinking they are foreign. This theory may explain why some women develop endometriosis and some do not: in healthy women, the immune system functions normally and helps to clear the pelvic cavity of any misplaced menstrual debris "regurgitated" through retrograde menstruation. In women with endometriosis, the impaired immune system is unable to perform this function. Instead, the immune system tags these wandering cells as "foreign" and reacts accordingly: the immune cells cause inflammation in the area, which leads to pain. Due to these immune cell abnormalities, the endometrial lesions are actually able to proliferate rather than being destroyed. This suggested auto-immune disease may also shed light on the tendency of endometriosis to appear alongside other diseases such as rheumatoid arthritis, diabetes, and hypothyroidism—autoimmune diseases, all. It's a link that will be explored more fully in a later chapter.

Another suggested cause of endometriosis has been, and continues to be, a controversial one—dioxin exposure. Dioxins, along with other similar chemicals, are found in pesticides, in some plastic products, and in bleached paper products. The Endometriosis Association (EA), an international organization devoted to providing support and catalyzing endometriosis research, has long argued that studies support the theory that dioxin exposure
can cause endometriosis—and, more alarming, that these dioxins are present in tampons and maxi pads. The EA supported research on a group of rhesus monkeys that had been exposed to dioxins and subsequently developed severe cases of endometriosis. The studies began in the early 1990s and are ongoing today, with the most recent report published in 2001. Other studies have seemed to demonstrate the opposite, that animals exposed to dioxins develop endometriosis at rates comparable to control groups. Yet the possibility of harm is strong enough that the Environmental Protection Agency (EPA) declared that one of dioxin’s effects is “a higher probability of experiencing endometriosis and the reduced ability to withstand an immunological challenge.” Nevertheless, the debate is fierce and ongoing, and unlikely to end soon. After all, a clinically controlled double-blind study—where half the women are purposefully exposed to dioxin and the other half are not—would be completely unethical. It’s going to be a long time before this question is settled.

Finally, genetics is likely a key player. Studies in Iceland, England, and the U.S. have traced the disease through families and are beginning to find that heredity does, indeed, play a role in endometriosis, a fact I can easily confirm. In August 2001, the UK-based Oxegene study joined forces with the Australian Gene CRC study (the two most extensive endometriosis-genetics research projects in the world) to form the International Endogene Study, which will ultimately contain data from more than 5,000 women with endometriosis, along with their family members. The evidence has begun to point to specific regions of the chromosomes, and now the task will be to determine which specific genes may be involved. What also remains unclear is whether a genetic hitch actually causes endometriosis or whether this hitch merely makes women more susceptible to other factors, such as retrograde
menstruation or that fundamental auto-immune disorder. Nevertheless, the progress made so far is heartening.

And yet, the only point made truly clear by these theories is that the origins of endometriosis—the roots of the disease’s story—are still remarkably unclear. But then, it is usually difficult—finding where things begin. Defining the line between normal and diseased. Pinpointing the cause of any effect.

My sister and I, after being diagnosed, both knew where to turn to find the origins of the disease in ourselves. For better or worse, we turned to our aunt and our mother.

“Most girls could wear Tampax and go swimming,” my aunt Lorrie, my mother’s only sister, said to me over the phone. “I could never do that. I could never wear Tampax. The heavy bleeding would shoot the sucker back out.”

I wasn’t sure how to respond to this, so I just let her keep talking. It was several months after my sister had been diagnosed, and I’d decided to begin researching more fully this disease that had crept into all of our lives. So I called my aunt in Arizona, and she was delighted to reminisce—vividly.

“Yeah, the cramps were incredibly painful—the kind where you just laid in bed and cried. I had nausea and vomiting, too. I mean, I could count on missing at least two days a month of school or work. And the bleeding was so heavy! When one of my girlfriends told me that periods were only supposed to last a few days, I told her, ‘You’re full of shit.’ I really didn’t believe her. I thought ten or twelve days was normal. My last one—before they did the hysterectomy—I bled for five months straight.”

She had her hysterectomy at the age of 26, after having her only child—Paul—at 21. The doctors told her she was lucky she’d been able to have a child at all, that she should have
never gotten pregnant or been able to carry a baby to term. But in a way, she had just been heeding the doctors’ advice.

“They told me to have a baby and it will go away,” she said. “Or they’d tell my mom that it was all in my head because that’s what my mom was feeding me—that it was supposed to hurt.”

One doctor, before the pregnancy, put her on birth-control pills. At the time, the only pills available contained estrogen and nothing else. My aunt said that she gained about 30 pounds and that the other side effects—migraines and nausea—were terrible. And, as doctors know now, estrogen-only pills were the worst idea imaginable: endometriosis feeds on estrogen, sops it up like Miracle-Gro.

She stopped taking the pills because of the side effects, and became pregnant. After she delivered, instead of the problem clearing up, as the doctors had promised, the symptoms became worse.

“I remember my first period after I had Paul,” she said. “I remember I was driving home with him from the park, and the pain was unbearable. When we got home, I couldn’t get my hands off the steering wheel because they were clenched so tight.”

She’s lucky she had a son; she’s never had to experience the overwhelming guilt of possibly contributing a hereditary disease to the next generation. My mother—who has never been dealt four aces in her life—was unlucky enough to have two daughters. And those two daughters were unlucky enough to inherit her wandering womb.

The inheritance even makes sense etymologically: the word *endometrium*, when broken down to its Greek roots, means “from within the womb.” The word for womb, *meter,*
comes from the Greek word for "mother" and this creates a secondary, though unintentional meaning: this disease may be "from within the mother" if it is, indeed, inherited.

I don’t blame my mother, though she often blames herself. Sometimes she’ll say, “Maybe I shouldn’t have had you girls and passed all this stuff on.” My feelings aren’t hurt when she says this. My sister and I often tease our parents that, considering their combined genetic backgrounds, they never should have reproduced. Besides, my mother had no way of knowing that her own problems would be inherited; after all, she’d been told all her life, up until her own laparoscopy at the age of 44 and hysterectomy at 45, that the severe cramps were all in her head or that the pain was normal.

Her own mother—my grandmother—didn’t have endometriosis, but she knew that my mom’s intense pain wasn’t normal. She knew that something was wrong, and she knew that something needed to be done. So she took my mom to doctors—"goddamn doctors," my mom grumbles now—who brushed them off without listening.

My mom’s only comforts all those years were cups of steaming tea, a heating pad, and a murmur of "poor baby" from my grandma. And now, it’s usually when my mom is getting my heating pad or making me a cup of tea, while I’m curled up in pain, when she’ll mutter, “Poor baby. If only I hadn’t passed this on to you girls.”

It is, perhaps, both the disease and the comfort that come from the mother. It is metrical, a necessary balancing of grief and compassion.

This disease is paradoxical, is an enigma. How could it crawl through my sister’s abdomen, spread its tentacles so far, without causing any symptoms? Why were my mother and aunt both able to have children when they both had the worst stage of the disease? If my grandmother didn’t have endometriosis, if it didn’t travel through her DNA into the fledgling
cells of my mother and my aunt, what was its source? Was endometriosis passed to them through their father’s side of the family, as recent research suggests is possible? Or should we blame the environment, or self-betrayal by the body?

Besides its possible origins, there are other, much-debated questions about this frustrating disease. Why does it take so long for women to be diagnosed? How does endometriosis infiltrate the pelvic cavity? Why haven’t we found a reliable treatment or, better still, a cure?

Endometriosis is one of the most common diseases on the planet—more common than AIDS and cancer—but it is also one of the most poorly understood. As I began to research the disease, I found a great deal of information—and a great deal of that information was contradictory, confusing, or completely inaccurate. Myths about sterility, frigidity, and hysteria persist, even in this rational, scientific era. It is difficult to cut through these falsehoods to get to the truth.

This much, however, I can say with confidence: endometriosis changes your life. Its destructive power has ironically pulled together not only my own family, but women from across the globe. These women, mothers and daughters together, sisters and aunts, have rallied, have worked towards a cure. They have pushed for the ultimate moment: when a doctor tells a girl’s family that she has Stage Four endometriosis, but does not speak of infertility, pain, the loss of motherhood, but instead says, “Don’t worry. That’s curable.”

This, then, is the story of a disease, of women, of pain, misunderstanding, patience, support, and renewal.
Saltines, Anaprox, and Blackberry Wine

August 2001

The worst pain I’ve ever experienced began at my grandma’s place, I remember, a few years ago—the familiar dull ache in my abdomen and lower back that foretold coming cramps. I tried to ignore the spasms, which were growing stronger by the minute, while we drove with my grandma to the drugstore to pick up a few things. She and my mom wandered the aisles as I headed for the feminine hygiene display, picked out the thickest, longest, most winged pads I could find, and paid for them. The cashier offered me a friendly “Have a nice day,” but my mind was somewhere else, trying to get far away from the waves of intense pain now racking my body, squeezing my entire abdomen into a state of unbearable tension. The cramps have never been this bad before, I thought. How odd. I know I took my Anaprox this morning.

I went back to the car and sprawled out in the backseat, trying desperately to find a position that would lessen the cramps. My mom and grandma soon joined me, and my mom, seeing my eyes clenched shut, knew immediately what was wrong. We dropped my grandma off, after she’d murmured a “poor baby” my way, and we headed for home.

That car ride, which probably lasted only fifteen minutes, was the longest of my life. The cramps became worse, which I hadn’t thought possible, and my stomach immediately clenched up in a spasm of nausea. Using little-kid logic, which was all I could manage, I told myself that everything would be better once we got home. But by the time we pulled into the driveway, my stomach was in full-scale rebellion. By some miracle I still don’t understand, I did make it to the bathroom, the hallway blurring past my eyes, before the retching began.
My mom followed close behind, kneeling next to me as both vomit and tears flowed into the toilet. Every stomach spasm brought with it a new wave of cramping, and all my mom could do was rub my back, hold my hair, and wait.

Finally I sat back on my heels, my clothes soaked through with sweat and my head pounding. The cramps had stopped, relieved somehow by my vomiting. My mom got me to my feet, into my pajamas, and into bed. As I settled in, the phone rang, and I heard my mother say, “Oh, hi, Lorrie. Meg’s sick.” A pause. “Yeah, her period.” After that I fell asleep.

The next morning I woke feeling sore from throwing up, but otherwise fine. I cautiously got up and even more cautiously made some tea and toast. I turned to the comics and felt a twinge. A sip of tea. A stronger twinge. Deciding to play it safe, I took my tea and toast back to bed with me, and added my battered heating pad, an old friend, to the mix. The warm tea, the warm heating pad, and the warm bed, however, didn’t stop the pain. I shut my eyes, breathed deeply, tried to focus on something else. Nausea set in. I ran for the bathroom again. My mother followed.

For one hopeful moment after this newest bout of retching, I thought that the cramps had stopped again as they had the day before. Then another wave hit, the strongest yet, and soon I was curled up on the floor of the bathroom, telling my mother shakily, “I can finally understand why that girl with endometriosis committed suicide.” I’d forgotten about her story, which I’d read in one of my mom’s Endometriosis Association newsletters, until that moment. And still, through that intense pain, I knew I was lucky: for some inexplicable reason, I lived this nightmare only occasionally. Some women, like the one who had killed herself, lived it every month.
I tried pacing, bent over double and clutching my stomach, but I then retreated to bed, shifting ceaselessly, the waves of cramps coming closer and closer together until I could no longer count the seconds in between. I curled onto my side, crying, my mother again rubbing my back, muttering words of comfort and despair.

"I can’t stand it," I cried. "I can’t stand it."

Eventually my mom left me for a moment, and returned with a glass of blackberry wine, a can of flat Coke, and a plate of saltines. I held the wine in one hand and the Coke in the other, taking sips of each, and nibbling at the bland crackers, as my mother sat in my room and drilled me on GRE vocabulary words in an attempt to take my mind off my suffering. I answered her for awhile—fluvial, trenchant, obdurate—until the wine made me fuzzy, and then I began to drift off. Immedicable.

My first period—back before I knew about blackberry wine—began on Halloween night, in 1992. I remember coming back from ringing doorbells, going to the bathroom, and staring at the dark red spot with utter disbelief.

Trick or treat.

Most girls, I suppose, when informing their mothers about this momentous occasion, receive a congratulatory “Welcome to Womanhood” speech. When I told my mother, her response was more along the lines of, “Oh dear.” I didn’t wonder at her lack of excitement; I’d seen enough of her suffering to know that “becoming a woman” was not necessarily something to celebrate. Still, though, I mused, things might be different for me. Perhaps my similarities to my mother ended with weak eyes and a passion for reading.

But within months, once my period had progressed from a single red spot to a full-fledged flow, the cramps began—different from any other pain I’d ever experienced, but
instantly recognizable from cycle to cycle: a squeezing, aching, heavy spasm that came and went in waves and ranged from my stomach to my sides all the way around to my back. My entire middle section felt as if it were swathed in a too-tight brace while my innards desperately wanted to escape from my abdomen and were determined to push their way out.

There are a few theories for what causes the intense pain.

Endometrial tissue produces hormones called prostaglandins. When these prostaglandins are in the endometrium, and the endometrium is where it’s supposed to be (inside the womb), the prostaglandins help to stimulate contractions of the uterus—which comes in handy during birth, as well as menstruation, when the endometrial cells need to be sloughed out of the body. But when the endometrium of endometrial implants produces prostaglandins, the hormones are released into the pelvic cavity and cause severe pain when they touch the tissues inside. Research suggests that these abdominal tissues have a low threshold for pain, and thus are more affected by the inflammation. This is why anti-inflammatory drugs, such as naproxen and ibuprofen, have offered some women relief from endometriosis pain.

Endometrial adhesions—implants that have adhered to the pelvic wall or to abdominal organs—may also cause pain, particularly when adhesions affect the bowel, ovaries, or uterus itself. When the adhesions are twisted or pulled during, for instance, a bowel movement, there may be sharp, shooting pains across the abdomen. Adhesions on the ovaries can lead to excruciating pain during ovulation. And adhesions on the bladder can cause bladder spasms and pain during urination.

Medications to ease this pain, then, are the first line of defense in endometriosis treatment. If anti-inflammatories fail, some women move to heavy pain-killers, desperate to
find relief from the tension. Unfortunately, these medications do nothing to stop the disease; they simply mask the symptoms. When these attempts at pain relief aren’t enough, women are usually told to try the birth control pill.

Using birth control pills to treat endometriosis actually makes a great deal of sense, biologically speaking. Birth control pills, when taken continuously (skipping over the week of placebo pills), place the body into a state of pseudo-pregnancy, altering the hormone levels within the body. Progesterone levels remain high, as they would during pregnancy, while estrogen levels remain low. The endometrial implants seen in endometriosis require estrogen to thrive, which is why the estrogen-only birth control pills my aunt once took made her endometriosis worse.

There are, however, a couple of problems with birth control pills as a treatment for endometriosis.

First, birth control pills are not easily tolerated by some women, a fact I can testify to. They can cause nausea, migraines, weight gain, and other such side effects, as well as more dangerous problems such as strokes and blood clots. While many side effects will decrease or disappear after several months of treatment, a woman suffering with endometriosis who has endured monthly pain for years may feel too desperate to endure even more physical hardships, especially if the side effects are particularly severe. When I began taking the pill and found I was simply adding more nausea and vomiting to my usual state, I lost my enthusiasm for the idea, especially when I learned that the pills would only keep the endometriosis at bay while I was taking them; they would have no long-term effect on the implants themselves once I stopped the treatment.
The other major problem with the birth control pill treatment has to do with the endometrial implants themselves. Researchers have discovered that these implants express high levels of an enzyme called aromatase, which synthesizes estrogen. The implications of this finding are discouraging, yet oddly amazing: endometrial implants are able to promote their own growth by synthesizing estrogen, so that they can continue to survive and spread even if the body’s natural level of estrogen is altered. These endometrial cells are wily, tenacious, stubborn. You almost have to admire their resourcefulness.

If birth control pills don’t work, the next step is the use of pseudo-menopausal drugs, according to the standard medical model. These drugs work on the same principle as birth control pills: dry up the source of estrogen and starve out the endometrial implants. But again, if endometrial implants can produce their own estrogen, cutting off the body’s natural estrogen would have little effect on the implants. This is why women who have had full hysterectomies can still suffer with symptoms of endometriosis: if the surgeon has missed even a tiny speck of endometrial tissue, it can survive on its own estrogen, or on supplemental estrogen from hormone replacement therapy, and continue to wreak havoc in the pelvic cavity.

Pseudo-menopausal drugs bring with them the usual host of menopausal problems, such as hot flashes and headaches, and they may also cause other side effects, depending on the type of drug: Danazol, which is chemically similar to testosterone, may cause problems that are related to the male hormone, such as acne, hair growth on the face, muscle cramps, and a decrease in breast size. GnRH (gonadotrophin-releasing hormone) agonists and antagonists, which work to inhibit the functions of the ovaries, can trigger depression or panic disorder, as well as bone loss.
Many women, frustrated with the inadequacy of the drugs available to treat endometriosis, have turned to alternative therapies, including acupuncture and acupressure, herbal remedies, and traditional Chinese medicine. These treatments have proved just as effective as standard medications, though doctors do not always embrace them as legitimate remedies.

My mother once read that blackberry wine could help to soothe menstrual cramps, so when the Anaprox came too late and the pain grew more intense, she would pour me a glass. The dark-red sweet viscous wine, it seemed, relaxed the pelvic muscles somehow, allowing them to stop their frantic spasms. When I later mentioned this to a gynecologist, he laughed, apparently skeptical of such “folk remedies.”

By the time I had my worst attack, the one that began at my grandma’s place, I was starting to lose hope. My current doctor had written me a prescription for the wrong form of Anaprox, something I didn’t realize until it was too late. I was still recovering from my latest round of birth control pills. None of the medications I’d tried had helped. And my pain, it seemed, was beginning earlier and earlier each month and lasting much longer into my cycle. It had been nine years since my period first began, nothing had changed for the better, and vague fears were beginning to settle in my mind. A laparoscopy remained a distant option, since surgery would be difficult to fit into my school schedule. It was also a step I was reluctant to undergo ever since my first gynecologist had explained the possible (though very unlikely) risks. I didn’t like the sound of a punctured bowel.

I could only hope that my scheduled appointment with another gynecologist, Dr. Anderson, would be fruitful. I went to his office (my mom in tow), sat down, and began the long wait.
“Melanie Griffith?” the nurse finally called out from the open doorway.

I sighed, placed my years-old Time magazine on the table next to my uncomfortable chair, and stood. I walked towards the nurse and she smiled grimly.

“Melanie Griffith?” she asked again.

“Actually, it’s Megan Griffiths,” I said. “Common mistake.”

She ignored me and turned away, saying, “Follow me.”

I followed her, and my mother followed me. We said nothing to the nurse. She didn’t exactly radiate warmth, and I was relieved. I didn’t feel much like talking. It had been a week since my two-day ordeal with cramps and nausea, and I still felt shaky.

The nurse led us into an exam room and then shut the door. She was Nurse Ratchett, a model of brisk efficiency.

“Age?” she said.

“Twenty-two,” I said.

“Are you in any pain right now?”

“No, not right now,” I said.

“How long have you had the problem?” she asked.

“Forever. Nine years, I guess,” I said, and I couldn’t help but sigh again. I had been through this history so many times, with so many nurses’ aids, with so many nurses who didn’t trust the nurses’ aids, with so many doctors who never read the nurses’ notes. I glanced at my mother, who was gazing at the floor.

The oral exam continued, the nurse taking copious notes. Finally, she finished and stood. I considered asking her how I did, but decided that she wouldn’t appreciate the joke.
“Doctor Anderson will be right in,” she said, leaving the room and shutting the door behind her.

My mother and I rolled our eyes. We’d heard that before. “Right in, my ass,” I said.

Forty-five minutes later, after we’d flipped through every magazine in the room and read every educational poster on the wall (who knew there were so many things that could go wrong with a woman’s reproductive organs?), there was a knock at the door. I sat up straight and wiped my hands on my jeans, hoping my palms weren’t too sweaty.

The door swung open and a thirty-something man walked through. He was holding my chart in one hand and offered me the other. “Megan?” he asked. I shook his hand, relieved that he had at least gotten my name right. He smiled at me, and I couldn’t help smiling back. Maybe he would be able to help me.

He sat on the round rolling exam stool and smiled at me again. I introduced my mother, and he smiled at her. Then he looked back at me with a smile.

“Now, what seems to be the problem?” he asked easily.

I knew then that he had not read the chart, had not even flipped through it to see what my symptoms were. But I continued to hope. I ran through my history again, twisting my hands on my lap. I felt the despair of the past two days lodged in my throat, tempting me to cry. I took a deep breath and forced myself to look up at him.

“I’m desperate,” I said.

He pulled his eyes away and glanced at my chart without opening it. Then he looked back at me and smiled. “Well, you certainly shouldn’t feel that way,” he said. “There are lots of things we can try that will help you.”
He began the standard sermon, one I’d heard a hundred times before. He mentioned pills I’d already tried, techniques that had failed. It was clear that he hadn’t listened to my history. He grew more and more patronizing, less and less willing to tolerate interruption.

I glanced at my mother again. Her eyes were focused on him, and they were venomous. I began to cry, out of utter frustration, and he ignored it.

He sent me off with a prescription for birth control pills and a smile.

I didn’t go back to that doctor again.

Being brushed off by doctors isn’t only frustrating—it’s dangerous. Because every time a doctor subtly suggests, through a sigh or a glance at the clock, that I am simply exaggerating the severity of my pain, or that I couldn’t possibly have already tried all the medications available, I wonder, for just a moment, if the doctor could be right. After all, these are people who have been to medical school, who have been trained for years and years, who, for all intents and purposes, ought to know a great deal more about the human body than I do. Perhaps, I think, perhaps I just have a low pain tolerance. Perhaps I’m weak. Perhaps the doctors are right to roll their eyes.

I’m not alone in this occurrence, or in my reaction. A November 2002 study done by the Endometriosis Association, which polled 968 women (ages 15 to 59) with chronic pain due to endometriosis, found that 40% of these women are told that they are exaggerating their pain. Of these women, 52% were told this by their gynecologist. And even a short visit to the various endometriosis message boards online immediately confirms this common experience—after listing her symptoms, usually including excruciating pain, and discussing all the pills she’s tried, the woman says she has been reassured by her doctor that there isn’t anything wrong. Her message then usually expresses some timid doubt over this diagnosis,
and often ends with, *Am I crazy?* She is quickly deluged with answers from other women with endometriosis, women who have learned how to push past the doctors’ dismissals and find answers.

Likewise, after every doctor’s visit, after every minute of doubt, and after every Anaprox that I swallow, I remind myself—*monthly pain is not normal.* I think back to those August days I spent curled up next to the toilet, shaking with pain. I think of the millions of women suffering worldwide, swallowing one hormonal concoction after another. I think back to my training as a biologist, my research into the molecular workings of endometriosis, and tell myself, over and over again, *You know as much as the doctors do.*

*And you are not crazy.*
I usually develop canker sores immediately before and during my periods.

I only noticed this a few years ago, and chalked it up at the time to an odd, irritating coincidence. But only months after I’d recognized this connection, the newest Endometriosis Association newsletter arrived. In the newsletter was a column featuring letters from women seeking further information, from other Association members, about a variety of topics. Halfway down was a query from a woman in Japan: “I get canker sores during my periods. Does this happen to anyone else? Please contact me.” Glad I’m not the only one, I thought, then filed the fact away as unimportant. Canker sores, after all, are just minor irritations—nothing to worry about.

Weeks later, pre-menstrual and with a new crop of canker sores, I went to the dentist for a routine cleaning. At the end of the appointment, I asked him if there was anything I could do to get rid of the canker sores.

“Not really,” he said. “I’ll give you some topical gel to relieve the soreness, but there’s not much else we can do.”

“What causes them?” I asked him as he searched through drawers for the gel. “I get them all the time.”

He turned back to me. “Well, it’s kind of unclear. Some people think it’s caused by a virus. Others think it’s because your body starts to attack its own cells, and that makes the area sore.” He went back to looking for the gel.

I stared at his back for a full ten seconds, in complete disbelief. Finally, I said slowly, “So what you’re saying is that this could be an autoimmune thing?”
“Yes,” he said, turning to me again, gel now in hand, seeming only mildly surprised that I’d known the technical term. “It could be an autoimmune thing.”

He handed me the gel and I left, too stunned to even thank him. My thoughts were slowly falling into place. *Mom has hypothyroidism, I have hypoglycemia—both autoimmune. Canker sores—possibly autoimmune. Endometriosis—linked to the immune system. Autoimmune diseases—often linked to endometriosis. Canker sores—endometriosis. Endometriosis—canker sores.*

*Canker sores?*

It’s a leap—a big leap. So far, there has been no documented evidence suggesting that those with endometriosis are more likely to get canker sores. Studies have shown that women are more likely to develop canker sores than men, and that women are more likely to get the sores immediately before menstruation, but plausible reasons have not been offered. Could endometriosis—an underlying autoimmune link—be the reason?

Perhaps the link between canker sores and endometriosis—if such a link exists—has not been studied because there are much more serious autoimmune links: endometriosis and lupus, endometriosis and rheumatoid arthritis, endometriosis and fibromyalgia. The list goes on—multiple sclerosis, hypothyroidism, diabetes. And on September 27, 2002, US researchers finally reported these links in *Human Reproduction*, demonstrating what women with endometriosis had suspected for years: that those with the illness are much more likely to suffer from autoimmune diseases, as well as immune hypersensitivity reactions including allergies, asthma, and eczema.

This is a topic that has scientists, as well as women with endometriosis, very excited. It’s a rare day in endometriosis research when a plausible, noticeable, practical connection is
discovered. It's an even rarer day when such a connection suggests possible treatments. The immune system—in all its complexity—may just be the link that's needed to treat, cure, or even prevent endometriosis.

When it's working correctly, the immune system is a marvelous thing. The immune cells are able to differentiate between "self" and "foreign" cells, sort out one type of foreign cell from another, mount a specific defense against specific foreign cells, rally other cells to join the fight, destroy the invader, and then remember how to kill the invader in case it ever shows up again. All of this happens through cell communication, which involves chemical and molecular signals. For instance, if a foreign cell (antigen) has entered the body, it will eventually bind to a particular T cell (a specific type of immune cell that recognizes foreign cells). That T cell, having recognized this antigen and been activated, will begin to divide in order to create more antigen-specific T cells. Also, once the T cell is activated, it will begin to produce a cell signaling molecule called interleukin-2 (IL-2). IL-2 urges the T cells to divide even faster, which in turn aids in the fight against the antigen. This is just a simple example of the complicated, web-like process used by the immune system.

Of course, anyone who works with computers would be quick to point out that the more complicated the system, the more chances there are for mistakes to occur. This seems to be precisely what happens to the immune system in women with endometriosis: parts of the process just break down.

To begin with, on a very basic level, these mistaken immune cells seem unable to tell the difference between "self" cells and "foreign" cells: this is the hallmark of an autoimmune disease such as hypothyroidism or rheumatoid arthritis. The immune cells come into contact with self cells—the endometrial cells that have somehow wandered up into the pelvis—and
panic. They begin to pump out antibodies, just as they would to fight a foreign antigen, and they send out signals to prepare for war. Inappropriate messages are sent to other immune cells, telling them to divide and conquer. The subsequent cell division provokes more messages of war. This activates the entire complex system of immunity, and shortly the woman’s pelvic cavity is the battleground in a war with no real enemy. All the classic symptoms of infection can occur: low-grade fever, inflammation, and pain—all the signs we assume are caused by foreign cells but are actually caused by our body’s fight against them. In the midst of this confusion, misfired signals are sent to the endometrial implants, with a message to proliferate. The correct signals—the ones telling the endometrial cells to disappear—are suppressed. And another part of this misdirected immune response seems to be the overproduction of prostaglandins, chemicals often linked to the pain experienced by women with endometriosis.

This is a persuasive and cohesive argument. It explains not only the monthly pain of endometriosis, but also the corresponding diseases, the chronic inflammation, and the spread of the endometrial implants. The idea, however, is not quite that simple. For instance, these theories suggest that a normal immune system would recognize wandering endometrial cells as self (misplaced, but self nonetheless) and clear them out of the pelvis without actually mounting an attack, thus avoiding the inflammation response. But other theories suggest that these misplaced cells would always be recognized as foreign—even in a woman without endometriosis—and that this immune response is the body’s way of cleaning up any retrograde menstruation that may have occurred. If this is the case, the theory argues, women with endometriosis don’t have a problem with autoimmunity; they have a problem with
suppressed immunity. Their immune system is too weak to take care of this misplaced menstrual debris.

What complicates the picture even more is the apparent connection between endometriosis and conditions caused by immune hypersensitivity, such as allergies, asthma, and eczema. These conditions are caused by a confused immune system yet again, only in this case, the immune system overreacts to harmless objects like dust and pollen instead of to self cells.

So which is it? Is the immune system hyperactive, or suppressed? Is it turning against itself, or unable to do so? There are arguments for both sides of these questions, again showing the paradoxical nature of endometriosis. Yet there is one fact, not yet discussed, that may bring these seemingly opposite theories together.

Yeast.

Women with endometriosis are more prone to yeast infections. This, along with the connection to autoimmune diseases, was simply an accepted fact among women with endometriosis for many years. The research community, however, has finally begun to turn accepted facts such as these into scientific breakthroughs.

The yeast *Candida albicans* has been clearly linked to allergic reactions, just as endometriosis has. In addition, *C. albicans* seems to induce the production of immune cell signaling molecules, such as interleukins, as well as certain prostaglandins, leading to an inflammatory response similar to what is seen in an autoimmune reaction. The most compelling evidence for a yeast-endometriosis connection, though, comes from what happens when *C. albicans* is absent: when women with endometriosis eliminated sources of the yeast from their diet, they found that their endometrial symptoms greatly subsided.
All of these correlations, as of right now, raise more questions than they answer. Is endometriosis an autoimmune disease, a state of chronic inflammation caused by a failure to differentiate between self cells and foreign cells? Or is the disease one of suppressed immunity, the inability to remove self cells from places where they don’t belong? How does hypersensitivity to harmless particles such as dust and pollen fit into the overall picture? Is \textit{C. albicans} the true culprit behind some of the most galling symptoms of endometriosis?

While working to answer these questions, scientists are also experimenting with ways to change the immune response in women with endometriosis. A drug called pentoxifylline works directly to alter the immune system by decreasing inflammation, which may greatly reduce the discomfort of the disease. Also being studied are the cytokines (cell signaling molecules, such as interleukins) that regulate the immune response. Since some of these cytokines tend to be overproduced in women with endometriosis, decreasing their numbers may dampen the immune response.

These are some of the most promising ideas in the field of endometriosis research today. No word yet, though, on the canker sores.
I am not a patient person. I had now lived with menstrual pain for more than nine years, and I was desperate for answers. I suspected endometriosis—my gut told me I had endometriosis—but there was only one way to be sure.

In May 2002, I met with a surgeon, specializing in endometriosis, at the Cleveland Clinic. We talked about my history, my symptoms, and my family background. He was knowledgeable and sympathetic, and I felt like asking him, Where have you been all my life?

“I very rarely do surgeries for women who have never had a laparoscopy before,” he said. “Most of the women I see have had seven, eight, nine surgeries already.”

“Oh,” I said. I paused a moment before speaking again. “Why do they have so many?”

“They’re desperate,” he replied. “They’re in so much pain, and the laparoscopy usually relieves it for a little while.”

I nodded. I’d heard that, but I’d also heard that laparoscopies often didn’t relieve pain at all.

He continued, saying, “Unfortunately, the more laparoscopies you’ve had, the less likely each one is to have much of an impact. But you should be fine.”

He pulled out some pamphlets and started explaining how a laparoscopy worked. I listened carefully, fitting in what he said with what I’d already read and researched on my own.

“It’s outpatient surgery—you’ll be in and out on the same day. We put you under general anesthesia, and the procedure generally lasts an hour or two. We make a small
incision on your abdomen and put some carbon dioxide gas inside your pelvic cavity. That gives us a little more room to work and helps us to see things more clearly." (I had a sudden strange image of myself floating upwards from the operating table like a balloon, and had to squelch the urge to laugh.) "Then we insert the laparoscope through another small incision, here," he said, pointing at a diagram. "The laparoscope is like a camera on a periscope—it lets us see inside your abdomen without us having to actually open you up. Then we'll make another incision or two depending on where we need to place other surgical instruments. Once we can see inside, we'll check out everything—your bladder, bowel, ovaries, uterus, fallopian tubes, everything. If we see any endometrial adhesions, we'll burn them out," he concluded, folding up the pamphlets. "Does that make sense?"

*Does it make sense that the laparoscopy may or may not help? Does it make sense that you may discover that I have only minimal Stage I endometriosis, even though I have all this pain? Does it make sense that a one-hour procedure could, possibly, end that pain? Does it make sense that, after years of research by a handful of dedicated individuals, this is still the best that science can do?*

"Guess so," I replied.

There was, and is, after all, no other choice. Endometriosis, as of right now, can only be diagnosed through surgery—typically, through a laparoscopy. There’s no quickie blood test, no easy “if this paper turns red” procedure. Even if the doctor detects something odd during a gynecological exam, the only way to know for sure if it’s endometriosis is to pull out the surgical tools. It’s surgery or uncertainty.

This is one reason why there is such a delay in diagnosis, and why endometriosis continues to be an under-diagnosed disease: surgery, even if it’s low-risk and minimally
invasive, carries with it certain risks that many women are unwilling to take. Laparoscopic surgeries carry the usual risk of a bad reaction to anesthesia, as well as possible damage to the bowel, bladder, or surrounding blood vessels by the laparoscopic instruments. These risks are miniscule, especially when compared to laparoscopy’s predecessor, the laparotomy (a full-incision, inpatient operation that requires several weeks for recuperation).

Laparoscopy requires only a few days’ recuperation time and usually doesn’t involve a hospital stay. Additionally, laparoscopy serves both as a diagnostic and a treatment tool.

The surgeon, upon diagnosing endometriosis, can either burn out or excise the endometrial implants, depending on their depth and location, and any endometrial adhesions that have formed can also be cut during surgery.

Some women decide against surgery because of an ironic fact: surgery to treat the implants and adhesions of endometriosis can sometimes cause post-surgical adhesions, leading to the same pain that brought them to surgery in the first place.

Laparoscopic surgery has mixed results, as the surgeon told me right away: some women experience immediate and permanent relief after the surgery, while others are completely unaffected. Those who are unaffected may turn to hysterectomy as a last resort.

One of the most prevalent myths about endometriosis is that it can be cured by hysterectomy. It does seem like common sense, though: if you remove the uterus, along with the estrogen-producing ovaries, surely the endometrial implants would soon die out.

To believe this is to underestimate this disease.

Endometriosis does not need estrogen from the ovaries, because it can synthesize its own. And even if the endometrial implants don’t pump out enough estrogen on their own, they can usually depend on the fact that the post-hysterectomy woman will be on hormone
replacement therapy (HRT), thanks to fears over osteoporosis and heart disease. HRT will provide the implants with a steady dose of estrogen.

This is not to say that hysterectomy can never cure a woman of endometriosis. Many women have experienced total relief after a complete hysterectomy, once they have dealt with the emotional and psychological issues wrapped up in the procedure.

But, as Mary Lou Ballweg, president and co-founder of the EA states very forcefully in *The Endometriosis Sourcebook*, “We’ve learned too many times, from the heartbreak of our members, that endometriosis is a stubborn, persistent disease. To leave traces of endometriosis behind . . . and think they will just disappear because the ovaries are gone is wishful thinking.”

Yet my mother has been told twice since her hysterectomy, when she told doctors of her pelvic pain, that it couldn’t possibly be due to endometriosis, because she’d had a complete hysterectomy.

In this age of open heart surgery, organ transplants, and gastric bypasses, it is sometimes difficult for people to imagine an illness for which no surgery is an absolute permanent cure.

So if surgery is not a completely successful treatment for endometriosis, why bother undergoing it? The answer, for many women, is a simple one: affirmation of their own experiences. An acknowledgment of their suffering.

This is all I was hoping for, when I finally had my laparoscopy. I woke up in the recovery room, trying to orient myself.

Cold and pain, and voices, floating beyond my hospital bed.

“And this young lady was cauterized for endometriosis, five incisions . . .”
Someone was just starting their shift, being filled in by someone else, I reasoned. How could the surgery be over already? How could I already be in the recovery room? Why was it so cold?

Eventually someone realized I was awake and asked me if I was in any pain. “Yes,” I muttered, my eyes still closed. “God, it feels just like cramps.”

“On a scale of one to ten, how bad is the pain?”

“I don’t know,” I moaned. I hated this question even when I was in a good mood; to have it asked now was unbearable. It hurt, it was pain, it was bad pain—what else could I say? I picked my lucky number. “I don’t know—seven.”

“Oh,” she said, her surprise evident. She fiddled with my IV bag.

“Can I have another blanket?” I asked, still shivering.

She piled on another blanket, and let me rest as the pain medicine started to kick in.

As the pain began to subside, the conversation I’d heard drifted back to me, and I realized its meaning.

I’d been cauterized for endometriosis. I had endometriosis. I’d been diagnosed.

My parents later joined me in the recovery room, where I was munching crackers with unabashed glee, finally free of the clear liquid diet. The nurses had given me some sort of anti-nausea drug, and my appetite was as strong as it had been that morning before the surgery.

My mom pulled out the pictures the surgeon had given them. “It’s here and here,” she said, pointing to small black dots, circled with pen, on the pictures. “It was on your ovaries and your bladder and somewhere on the walls. Stage Two,” she said.
“And the worst is Stage Four,” I finished. “Unbelievable.” I ran my fingers over the black spots, amazed that such tiny lesions could cause so much pain.

“He said he’ll fill you in on details at your post-op appointment,” my dad added.

I nodded and continued eating my crackers. “It was on my bladder? Maybe that’s why I’ve got to pee all the time,” I said.

My dad laughed. “Yeah, Pees-a-Lot,” he said, using the nickname he’d given me as we’d pulled into yet another rest stop on a family vacation.

It was ironic then, and more than a little irritating, that I couldn’t pee after the surgery. Maybe it was due to the catheter, maybe it was the cauterization of endometriosis on my bladder, but, without warning from the nurses, I couldn’t urinate. I sat on the toilet in the recovery room for many long minutes, willing just a bit to trickle out, knowing the nurse was standing impatiently outside. I sat later on the toilet at home, thinking about the astronomer Tycho Brahe who had died of an exploded bladder, and I couldn’t pee. It took days for my normal abilities to kick back in.

I thought a lot, after the surgery. I thought about the diagnosis. I thought about the pain in the recovery room, and wondered if I’d ever have that kind of pain again. I thought about those nine years of doubt. I thought about my mom, as she sat on the chair next to my bed before they took me in for surgery. She’d looked at me and tried to smile.

The next few days, once I’d recovered from the peeing problem, were better than I’d expected. There was pain, mostly when standing up or sitting down, but it was pain that felt like recovery, like a healthy stretching, that foretold a possible end to suffering. I was hopeful. I showed my pictures to anyone who would look.

“Look!” I said. “That’s my ovary! Look at the egg popping out there!”
People nodded politely, averted their eyes, and asked if I was feeling better.

Two months later, my sister was waking up in the cold, in pain, hearing that she’d been cauterized for Stage Four endometriosis.
In a college class I took about myths several years ago, we watched a video called *Goddess Remembered.* It talked of women’s monthly connection to the earth, their communion with nature, their fertility and potential for creation and creativity. Women, it said, are reclaiming the idea of the goddess, an Earth Mother who lives in harmony with snakes, and showers the world with crops. The idea is an ancient one, stretching back to Sumer and the Greek world; the Sumerians claimed Inanna as their Earth goddess, while the Greeks celebrated Demeter as the goddess of fertility, agriculture, the woman’s life cycle, the mother-daughter relationship, life and death. Demeter was the corn goddess, who caused all good things to grow. She was a mother.

Because of the pain that had, for me, become synonymous with my own sense of being female, I had never celebrated my womanhood. I had never considered the mystical connection between myself and the women who came before me, between myself and Eve, the seeker of knowledge, the first mother, between myself and Demeter, the woman who searched heaven and hell for her lost daughter Persephone. Inanna tells her lover Dumuzi that her fields are fertile and waiting to be plowed. Mine lay in fallow, perhaps never meant to bear life.

I have shoved the thought away, never confronted the very real possibility that I might remain childless, that I might be unable to fulfill my possible role of creator, because of this disease, because this disease had crept into my fortress and destroyed the treasures within. It had wandered, uninvited, into sacred meadows and plucked their plants in mid-bloom.
I have tried not to consider the idea that this disease has affected not only my own life, but the potential lives within, those whose possibilities might turn out to be impossible.

How appropriate, I thought as I watched the video, that the Earth goddess is usually in the company of snakes. Snakes shed their skin, leave behind past mistakes in favor of newer, fresher tissue. Women shed their endometrium to create a new chance, every month, for life to take root and grow. I couldn’t shed my past completely. The old constantly mingled with the new, making an environment hostile to new life.

The most recent estimate is that about one-third of women with endometriosis have infertility—two or three times the normal rate; likewise, about one-third of women with infertility have endometriosis. Some women only discover they have endometriosis because they are unable to conceive and seek an explanation for the problem. And as with nearly everything concerning this disease, the reasons for this infertility are still unclear. Numerous theories exist; as with the possible causes of endometriosis, it’s likely that any or all of these factors could be involved in individual cases.

One of the most obvious possible causes of infertility is the distortion of the reproductive organs in a woman with endometriosis. Endometrial implants or adhesions might block the fallopian tubes or take over the ovaries, thus disrupting the release of eggs or their fertilization. Laparoscopy can sometimes solve this problem by clearing the tubes or ovaries of endometrial growths, allowing eggs to pass unhindered out of the ovaries and into the fallopian tubes. If these obstacles are removed, fertilization may be able to take place as normal.

The other suggested causes of infertility in women with endometriosis are more difficult to solve. Women with the disease have been found to produce lower than normal
levels of certain integrins (cell adhesion molecules), which are necessary for embryo implantation in the womb. Higher levels of immune system chemicals, such as prostaglandins, are also found near endometrial implants, creating a peritoneal environment that is hostile to both eggs and sperm and thus disrupting fertilization. Hormonal imbalances due to endometriosis have also been blamed for both ovary malfunctions (such as failing to release an egg) and uterine lining abnormalities, again affecting both fertilization and embryo implantation in the womb.

There are options for women with these problems. Infertility treatments include hyperstimulating the ovaries in order to increase the number of eggs that are released, injecting sperm directly into the uterus so that it doesn’t come into contact with any hostile chemicals, and utilizing in vitro fertilization (IVF). The effectiveness of these treatments in women with endometriosis is still unclear, since some studies suggest that the success rates in women with endometriosis are lower, while others suggest that there is no difference.

Regardless of the treatments’ effectiveness, women (and their husbands) will invariably need to deal with the emotional issues brought up by both the infertility itself and how it is treated. Women often have feelings of anger, guilt, and inadequacy, along with a great sense of loss. Relentlessly pursuing fertility treatments has also strained many a marriage. In addition to confronting the emotional trials of infertility, couples may also feel the strain of the financial aspect of infertility, since treatments are usually not covered by insurance and can drain a couple’s budget.

Another practical complication concerning fertility treatments deals with treating the endometriosis itself. Most women who suspect endometriosis, at some point, are put on birth control pills to hormonally suppress the endometrial implants. It can take several months for
the effects of the pills to wear off, but at the same time, the endometrial implants can quickly begin to re-assert themselves. Timing is truly everything. Additionally, women attempting to get pregnant are advised to avoid using non-steroidal anti-inflammatories (such as Anaprox or ibuprofen) during mid-cycle, because these drugs have been found to interfere with ovulation. So another irony is revealed: the very pills that women take to treat their endometriosis (a disease that can interfere with pregnancy) are the exact pills that women shouldn’t take if they’re trying to get pregnant.

I have not thought much about my fertility, or lack thereof, because my life right now isn’t conducive to having children. I have, though, spent some time considering my sister.

My sister had told everyone, from the time she was little, that she didn’t want to have children. “I’m a teacher,” she’d say. “I’m around kids all day. Why should I have to deal with them when I go home?”

Secretly, I never believed her. I suspected that she knew, on some bone-deep level, that she’d never be able to have kids, and her bold condemnation of the idea was her way of accepting the impossible. When the surgeon informed us of the Stage Four endometriosis that had taken over her abdomen, I was glad that she’d (supposedly) decided against having children. *There’s no way she’d ever be able to conceive,* I thought. *And even if she did, who knows if she’d be able to carry the baby to term?* Her body was in terrible shape, and I couldn’t imagine how she could safely survive a pregnancy. It was a miracle that our mother and aunt had been able to have kids; I decided it was probably better if we didn’t try to push our luck. I was in no position yet to be making these choices, but I approved of hers.

It was just a couple years later when my sister got married, started talking about buying a house, and said to me those heart-stopping words: “I think I want to have kids.”
It was selfish, and cynical, and horrible, but all I could think was, *Please don't try.* It *just wouldn't work.* Aloud, I said, "You could always adopt."

There was a long silence before she replied, "Yeah."

We didn’t mention what was hanging between us: it’s entirely possible that the choice of our own children has been taken away from us both.

That we have been denied a vital part of our womanhood.
I’ve never been one to back away from a good (or even bad) pun. It must be genetic: my mother passed on endometriosis and compassion, my father passed on green eyes and punning. So, when cramps hit, I always remind myself that this pain won’t last forever, that every period, without fail, must come to an end. After all, unlike commas and dashes, periods are all about finality. And if a period has to end, so does endometriosis.

And the end may soon be in sight, thanks to a recent increase in two things that have long eluded endometriosis sufferers: awareness of the disease and sound scientific research.

On October 1, 2002, something truly remarkable happened: endometriosis became not only an official disease, but also a political cause. The House of Representatives passed Concurrent Resolution 291, which stated:

Whereas an estimated 10 to 20 percent of American women of childbearing age have endometriosis;

Whereas endometriosis is a poorly understood disease and can strike women of any socioeconomic class, age, or race;

Whereas the disease can affect a woman’s ability to work, ability to reproduce, and relationships with her mate, children, and everyone around her;

Whereas infertility occurs in about 30 to 40% of women with endometriosis;

Whereas the cause of endometriosis is unknown;

Whereas the disease can only be definitively diagnosed through gynecologic surgery;
Whereas studies have shown that the average delay in actual diagnosis is
more than nine years; and

Whereas there is no definitive cure for endometriosis: Now, therefore be it
Resolved by the House of Representatives (the Senate concurring), that the
Congress:

1. strongly supports efforts to raise public awareness of endometriosis
   throughout the medical and lay communities; and

2. recognizes the need for better support of patients with endometriosis, the
   need for physicians to better understand the disease, the need for more
   effective treatments, and ultimately, the need for a cure.

H. Con. Res. 291 was introduced in December 2001 by Rep. Howard McKeon of Santa
Clarita. In a brief press release about the resolution, McKeon said, “I had never heard of
endometriosis until a constituent, Mary Prenger, brought it to my attention.” Prenger, who is
the leader of the ERC’s Legislative Awareness Committee, said that this is not an uncommon
response. “In fact,” she said in the ERC’s jubilant report of the bill’s passage, “in
approaching various Representatives to garner support for H. Con. Res. 291, we learned that
many of them were unfamiliar with the disease until the ERC brought it to their attention.”

The ERC, accompanied by the cheering of women worldwide, managed to introduce
an entire Congress to the concept of endometriosis. In one fell swoop, they pushed forward
their goals of education, research, and support.

Long before Congress decided endometriosis was worth recognizing, the EA had
been working steadily to push endometriosis into the public eye. They convinced “Dear
Abby” to run an entire column about endometriosis. They declared that March was
Endometriosis Awareness Month. They supported research into the possible environmental causes of endometriosis. They held workshops, symposiums, and conferences. They raised money. They wrote books. They sent out newsletters, filled with the most recent discoveries and tips. They worked with universities and medical centers to create research programs that focused on aspects of the disease. They held the first Endometriosis Walk for Awareness in Washington, D.C. in 2000.

One of the best aspects of the EA is its widespread network of support. The organization is active in 66 different countries, and its Internet bulletin boards, along with its printed newsletters, contain messages from women in Sweden, England, Germany, Canada, France. Support groups meet around the U.S., and around the world, on a regular basis. The Internet has exploded with these kinds of endometriosis support systems, offered by a wide variety of groups. The ERC, The EA, and Endozone (sponsored by obgyn.net) all offer current, helpful information about symptoms, treatments, research, and tips from other endometriosis sufferers. There are endometriosis listservs, endometriosis discussion boards, endometriosis chat rooms, endometriosis webpages, endometriosis FAQs, and endometriosis e-mail groups. I signed up for the ERC’s support group listserv and began receiving daily e-mails containing postings from other women in the group. These women’s messages were incredible in their depth and courage. They discussed which treatments were working and which had nasty side effects, they soothed women anxious about their first laparoscopy, they talked about fibromyalgia and infertility and pain in their own lives, they offered compassion. These women have formed their own support systems.

It is because of the tireless work of these organizations, and the women who run them, that endometriosis research—into causes, symptoms, and possible cures—has gathered
strength and speed over the past few years. Numerous recent studies have shown the promise of cutting off angiogenesis—the formation of new blood vessels—in the endometrial tissue. Aromatase inhibitors—drugs that shut off the action of the estrogen-producing enzyme aromatase—are being tested with promising results. And surgical techniques to remove the endometrial adhesions continue to improve, including the creation of an anti-adhesion gel that is smeared into the patient’s peritoneal cavity during surgery, creating a sort of Teflon non-stick surface. Perhaps the most exciting development comes from the company Procrea BioSciences, Inc., which announced at the most recent World Congress on Endometriosis that its new, noninvasive diagnostic test for endometriosis—requiring only a blood draw and an endometrial biopsy—has been approved for use in Quebec and is expected to be approved in the United State soon. A non-surgical diagnostic tool would likely cut down on the delay between the onset of symptoms and the official diagnosis.

Unfortunately, much of this good research can be overwhelmed by studies that rely on less sound science, but are more widely publicized. For instance, last year my sister mentioned an article that she’d read in Cosmo. “It says that having orgasms and wearing tampons can prevent endometriosis!” she said.

I was incredulous. How could tampons prevent endometriosis? For years, research supported by the EA had connected tampon use to dioxin exposure—one of the proposed causes of endometriosis.

I was also intrigued. Was Cosmo actually onto something with this orgasm stuff, or was it just another way to move magazines?

So I turned to the Internet, one of the best places to find information about endometriosis, and directly found the answers to my questions at the ERC website.
The *Cosmo* article, it turned out, was based on a study published in the *Journal of Gynecologic and Obstetric Investigation*, and then reported in the *New York Times* on June 4, 2002. The study was led by Dr. Harvey Kliman of Yale, who stated that orgasms seemed to move the menstrual debris out of the body, and that tampons seem to “act like a wick.”

The ERC had a different take on the results, which were based on a survey of 2000 women. And Heather Guidone (Director of Operations) and Michelle Marvel (Founder and Executive Director) of the ERC didn’t hesitate to state their displeasure in a rebuttal letter they sent to the journal:

> Women and adolescents with this disease are already acutely aware of the cavalier attitude with which endometriosis is regarded—as a largely insignificant condition shrouded in myths and misinformation; it is viewed as “a disease of primarily middle aged career women; it can be cured by hysterectomy/pregnancy; it does not generally cause pain;” etc. Then comes the latest hindrance—news that endometriosis can be helped—and perhaps even prevented, some would interpret—by such simple measures as having intercourse during menses and/or wearing tampons.

Guidone and Marvel then dissected the flaws in Dr. Kliman’s study, piece by piece. First, they said, the study was based on Sampson’s Theory of Retrograde Menstruation (first proposed in 1927), a theory that practitioners clung to until they realized they could not use it to explain how endometrial adhesions appeared in places other than the peritoneal cavity; retrograde menstruation also could not explain the strange, albeit rare, appearance of endometriosis in men. Perhaps, suggested the ERC, Kliman’s work would have been more credible if he’d included references to research more recent than 1996.
Second, Kliman failed to address the harmful role of dioxins in tampon use, a phenomenon that, while still controversial, is accepted as a probable risk by most of the scientific community. Guidone and Marvel pointed out that a 1999 study had actually suggested that a reduction in tampon use would lower the incidence of endometriosis.

But most important, perhaps, was Kliman’s failure to recognize the “chicken or the egg” problem.

Women who use tampons and have orgasms during menstruation are less likely to have endometriosis, Kliman claimed.

But the ERC pointed out what may already be obvious to women with endometriosis:

All other evidence notwithstanding, this study fails to address 2 key facts known to be true by those who understand this disease the best—the women who live with the illness every day. Simply put, many women with endometriosis simply cannot bear the pain caused by tampon insertion and wearing. As for sex: painful intercourse is a hallmark symptom of endometriosis.

It’s not that women without endometriosis are saved from the disease because they’re using tampons and having sex; women without endometriosis don’t have endometriosis, and so are able to use tampons and have sex. Or as Guidone and Marvel put it, “Many [who read the article] will likely not even consider that the women who engage in such practices do so simply because they can—yet many with endometriosis cannot.”

The ERC closed their letter:

It is our strong opinion that advising the female public, particularly those already diagnosed with endometriosis, to subject themselves to painful (and in certain cases, potentially dangerous) activities borders on the side of negligence.
The ERC's letter was eventually printed in *Journal of Gynecologic and Obstetric Investigation*, along with a rebuttal by Kliman.

I've found it difficult to remain hopeful when studies such as Kliman's are the ones that make the headlines. But hope and compassion have popped up in strange places.

I see hope in my diagnosis, and in the well-meaning friends and family members who have asked me if the laparoscopy helped.

Yes. It's helped. I haven't thrown up from painful cramps since I had the surgery.

It's true that I still have medium-grade pain during my period, and that sometimes this turns into bad pain, but it usually doesn't last. This may seem like a small improvement, but with endometriosis you have to take what you can get.

And I'll admit it. When I woke up in recovery and heard the nurse say I'd been treated for endometrial lesions, I was relieved. Excited. Happy, even. Because this meant there was really a problem, a physiological problem, incurable and misunderstood, yes, but real and tangible. The years of guesswork and misdiagnosis had ended. In my still torpid state, I smiled and thought back to a parody someone had written for an Endometriosis Association newsletter: supercalafragalistic endo-diagnosis. I'd been diagnosed. My symptoms now fell under a name. It would no longer be, "I think I might have endometriosis." From now on, it would be strong and clear.

I've been diagnosed. I have endometriosis.

Whenever I tell someone I have the disease, they invariably respond, "Oh, so do I!" or "So does my sister!" or "So does my best friend!" In one of my graduate school classes last year, I was discussing endometriosis with another class member, Karen, who told me her sister has the disease. She inquired into my pain level.
“It gets bad occasionally,” I said, “but not nearly as often as it used to.”

“Yoga,” Karen responded. “Have you tried yoga?”

I opened my mouth to reply, but before I could say a word, Karen had dropped to the ground, partly unzipped the fly of her jeans, and lain on the floor of the classroom, her legs bent frog-like up against the wall, her feet touching and hands relaxed at her sides.

“This is a good yoga position to try,” she said, looking up at me from the floor. “It relaxes the pelvic muscles.” She took a couple of slow breaths, showing me how to breathe deeply into the pain. She opened her eyes to glance at me again. “If you ever want me to show you some more yoga positions to try, you can call me.”

I nodded and offered her some feeble thanks. I looked away, allowed my eyes to blur. I was near tears and couldn’t understand why.

The next time I had bad pain, I tried Karen’s yoga position. I was much less graceful getting into the stance than she had been, but once there, I could feel the relaxation she’d mentioned. I breathed deeply, over and over again, until I felt the pain subside somewhat.

I silently said a few words of thanks and hope to Karen, to my mother, to God. I asked for strength and patience.

My uterus has wanderlust, I guess. It wants to investigate areas beyond its own domain, and so has sent scout cells to explore the wonders of my ovaries and bladder. Apparently, these cells liked the new terrain well enough to drop roots and start families.

I almost feel bad that these adventurous little adhesions were burned out during the laparoscopy.
This sounds ridiculous, I know. But my molecular biology courses in college made me much more respectful of cells and their inner workings. The process of DNA replication stunned me with its simplicity. The sodium-potassium pump reaffirmed my belief in God.

And these endometrial cells are no different. They are tenacious and stubborn. If you take away their estrogen, they will make their own. If you burn them out in surgery, they will pop up somewhere else. You have to admire their resourcefulness.

So when I saw the pictures from the laparoscopy, with the black spots on my ovaries and peritoneal wall circled, I felt strangely torn. Yes, I hate endometriosis. Yes, it makes my life miserable. Yes, it may make me infertile. But it’s a pioneer. It’s tough. It’s resilient. It’s nearly unstoppable.

I hope the researchers working on this disease have the same respect for it.

It’s a formidable enemy. But every enemy has its weakness.

The weakness may lie in its genetic expression, or in its tendency to infiltrate new tissue. Perhaps it will be beaten by cutting off its blood supply or its source of estrogen, or even its ability to make its own estrogen. Maybe researchers will stop it by deciphering its relationship with the immune system or by figuring out how to reverse the harmful effects of dioxin exposure.

Until then, women with endometriosis must rely on their own strength and on each other. They must have faith that the disease will be cured. They must fight for more research and better awareness of the problem.

We must be as tenacious as the disease is. Period.
Notes on Sources

*Meter: Origins*

The Center for Fertility and Reproductive Endocrinology at Virginia Mason Medical Center claims that, if your mother and sister have endometriosis, you are ten times more likely to develop the disease (2001). This number is likely to fluctuate wildly as more and more research is done on the possible heritability of endometriosis.

Perhaps the most difficult source to cite is one concerning the numbers of women affected by endometriosis, because the numbers vary drastically from source to source. Most sources put the number at somewhere between 5% and 15% worldwide, though the article “Looking for a better way to manage endometriosis-related pain” in *Contemporary OB/GYN*, April 2003 states that “nearly one of every two women may now be affected” by endometriosis. It’s difficult to say whether endometriosis is actually becoming more common or simply whether more women are being diagnosed, but regardless, endometriosis is clearly one of the most common diseases on the planet, more common, as Endometriosis.org (affiliated with the ERC) states, than AIDS or cancer. The latest EA book (2004), called *Endometriosis: The Complete Reference for Taking Charge of Your Health*, contains the most recent estimate of 89 million women worldwide.

Vernon all provide excellent summaries of the theories regarding the causes of endometriosis, as well as citations for the original studies.

The EA, the National Institute of Child Health and Human Development in Bethesda, and the School of Public Health and Health Services at George Washington University carried out the 2002 study which demonstrated the co-occurrence of endometriosis and other diseases such as rheumatoid arthritis, fibromyalgia, and allergies in women.

Further information on the International Endogene Study can be found at either the OXEGENE website (http://www.medicine.ox.ac.uk/ndog/oxegene/oxegene.htm) or the Australian Gene CRC website (http://www.genecrc.org/index.htm).

Saltines, Anaprox, and Blackberry Wine

Again, the 1999 book *Endometriosis: A Key to Healing Through Nutrition* by Mills and Vernon clearly discusses the pain process in endometriosis, as does the ERC's 2002 report "Understanding Endometriosis: Past, Present, and Future."

The EA website, ERC website (http://www.endocenter.org/), and Endozone.org (sponsored by Obgyn.net) all contain updated, concise, and understandable explanations of the effects of endometriosis treatments, along with the stories of women who have been struggling with the disease and with the reactions of doctors.

Auto-Destruct

Any medical text, from the National Institutes of Health to webmd.com, contains the odd information that canker sores are more common in women than in men, and that women are more likely to develop canker sores pre-menstrually.

Information on the relationship between endometriosis and the immune system, as well as possible connections with *C. albicans*, is discussed in depth in the EA’s 2004 *Endometriosis: The Complete Reference for Taking Charge of Your Health* and in the 1999 book *Endometriosis: A Key to Healing Through Nutrition* by Mills and Vernon.

Peter Parham’s 2000 text *The Immune System* serves as an excellent introduction to the complexities of the immune response. The book also features chapters focused specifically on autoimmune diseases and immune hypersensitivity reactions.

Cavity Search

The EA rules the field on surgery information. Their 2004 book and their 1995 book *The Endometriosis Sourcebook* contain clear, thorough explorations of what laparoscopies and hysterectomies entail, as well as the possible outcomes of each procedure. Another good
source in this area is Robert H. Phillips’ and Glenda Motta’s 2000 book *Coping with Endometriosis: Sound, Compassionate Advice for Alleviating the Challenges of this Chronic Disorder.*

**Demeter**

The EA’s 2004 book again proves a useful source for infertility information, along with Mills’ and Vernon’s 1999 *Endometriosis: A Key to Healing Through Nutrition.*

The American Infertility Association’s website (http://www.americaninfertility.org/) has a vast amount of helpful statistics and practical information.

Phillips’ and Motta’s 2000 *Coping with Endometriosis* delves deeply into the emotional and psychological aspects of endometriosis, including the emotional process of fertility treatments and, eventually, pregnancy.

**Period**

The ERC website discusses, in depth, the passage of H. Con. Res. 291, as well as the steps taken to achieve this milestone. There are links, as well, to similar resolutions passed by specific state legislative bodies.

The EA’s 2004 book contains the speech given by Mary Lou Ballweg on the day of the first Endometriosis Walk for Awareness in Washington, D.C.
The Endozone message boards and ERC listservs provide valuable insight and support from and for women with endometriosis. Additionally, the ERC and EA run support groups both around the country and around the globe; local addresses can be obtained from the websites.

The 2004 EA book devotes many pages to the newest developments in endometriosis research. The EA and ERC websites also have “news” links that will display the latest research results.

The ERC website contains links to the Cosmo article, the original research done by Kliman, and the ERC rebuttal letter.