

**UNITED STATES DISTRICT COURT  
NORTHERN DISTRICT OF ILLINOIS  
EASTERN DIVISION**

<b>INNOCENT LIKONGA KASONGO,</b>	)	
<b>Individually and as Special Administrator of</b>	)	
<b>the Estate of JACQUELINE MAKOMBE,</b>	)	
<b>Deceased,</b>	)	
	)	
<b>Plaintiff,</b>	)	
	)	
<b>v.</b>	)	<b>No. 04 C 4901</b>
	)	
<b>UNITED STATES OF AMERICA,</b>	)	<b>Judge Rebecca R. Pallmeyer</b>
	)	
<b>Defendant.</b>	)	

**MEMORANDUM OPINION AND ORDER**

Jacqueline Makombe, her husband Innocent Kasongo, and their three children emigrated to the United States after enduring civil war and ethnic strife in central Africa. Raped by soldiers in 1998 during the Second Congo War, Ms. Makombe contracted HIV and subsequently developed AIDS. After arriving in Chicago in July 2000, Ms. Makombe received treatment at Chicago Health Outreach Clinic (the "Clinic"), a federally-funded clinic that provides medical care and social services to HIV-positive refugees and their families. Ms. Makombe's treatment included the AIDS drug Zerit. A rare but known side effect of Zerit is the often-fatal condition lactic acidosis. Although Ms. Makombe developed symptoms of lactic acidosis beginning in August 2001, medical staff at the Clinic did not diagnose the condition until October 22, 2001. By that time, the lactic acidosis was irreversible, and on October 24, 2001, Ms. Makombe died.

Mr. Kasongo, individually and on behalf of his children and his wife's estate, brings this action against the Clinic pursuant to the Federal Tort Claims Act, 28 U.S.C. § 1346(b)(1), asserting claims for negligence under the Illinois wrongful death statute, 740 ILCS 180/1 *et seq.*, and under the Illinois survival statute, 755 ILCS 5/27-6. Plaintiff contends that the Clinic and its employees breached the applicable standard of care by failing to timely diagnose lactic acidosis, and that that failure proximately caused Ms. Makombe's death. The court conducted a bench trial in December

2006 and, for the reasons set forth below, now finds in Plaintiff's favor. The court awards Plaintiff \$3.5 million in damages for the wrongful death claim, and \$1 million in survival damages.

### **BACKGROUND**<sup>1</sup>

The basic facts of this case are undisputed. Beginning on July 24, 2000, Ms. Makombe received treatment for HIV and AIDS at the Clinic. (UF ¶ 4.) That treatment included the antiretroviral drug Zerit, which is the brand name for stavudine. (*Id.* ¶¶ 22-23.) On October 22, 2001, the Clinic referred Ms. Makombe to Weiss Memorial Hospital ("Weiss"), where she was diagnosed with lactic acidosis. (*Id.* ¶¶ 25-26.) It is undisputed that the lactic acidosis was caused by Zerit, and that she died as a result of lactic acidosis on October 24, 2001. (*Id.* ¶¶ 27-28.) The dispositive issue in this case, for purposes of liability, is whether the standard of care required the Clinic to have diagnosed lactic acidosis prior to October 22, 2001.

Plaintiff Innocent Kasongo, Ms. Makombe's surviving spouse, is the appointed, qualified and acting personal representative of Ms. Makombe's estate, and has been authorized by Illinois courts to bring this action on behalf of their children Moises Kasongo, Ange Kasongo, and Sara Kasongo. (*Id.* ¶¶ 7-8.) The federally-funded Clinic, and all its employees who provided care and treatment to Ms. Makombe, have been deemed by the Department of Health and Human Services ("DHHS") to be employees of the United States for purposes of the Federal Tort Claims Act, 28 U.S.C § 1346(b)(1). (UF ¶¶ 5-6, 9-12.)

Clinic care providers and employees Dr. Marcia Katz, Mary Tornabene, Tamara Falk, Celine Boers, Mary Lynn Everson, and Heidi Nelson testified as adverse witnesses during Plaintiff's case-

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<sup>1</sup> The court presents findings of fact and conclusions of law pursuant to Rule 52(a) of the Federal Rules of Civil Procedure. Facts are drawn from the pretrial order and from evidence presented at trial. The parties' Stipulation of All Uncontested Facts, Schedule 2(a) to Final Pretrial Order, is cited here as "UF ¶ \_\_\_\_." As the trial transcript is available only in rough form, the court cites that transcript by the name of the witness and the date of testimony, as "[last name of witness] 12/\_\_/06." The parties' trial exhibits are cited as "Pl.'s Ex. \_\_\_\_" and "Def.'s Ex. \_\_\_\_." The court cites Jacqueline Makombe's medical chart by Bates numbers.

in-chief. Mr. Kasongo testified as well. Dr. Larry Rumans, Dr. Richard Novak, and Sheldon Fields testified as medical experts for Plaintiff, and Gerald Richard testified as an expert in the area of document examination. Dr. Harold Kessler and Bradford Farrington testified as medical experts on behalf of the United States.

**A. The Parties**

**1. Jacqueline Makombe and Her Family**

Ms. Makombe was born in what is now known as the Democratic Republic of the Congo on April 24, 1962. Although she was raised Congolese, her parents, who had emigrated from Rwanda in 1959, were of Tutsi descent. Mr. Kasongo was also born in the Congo and is Congolese. Mr. Kasongo and Ms. Makombe were married in 1987 in a folk ceremony, and renewed their vows in a civil ceremony in 1994. They settled in Kinshasa, the capital of the Congo, in 1989. Their three children are Moises, who was born on April 13, 1991; Ange, born on November 19, 1993; and Sara, born on May 22, 1996. (Kasongo 12/13/06.)

Mr. Kasongo holds a diploma in economics and finance, and also received a diploma in “expert detective work” to assist with an accounting practice in which he investigated money laundering. His native language is French, and although he is able to communicate in English, he testified at trial through an interpreter. (*Id.*)

At trial, Mr. Kasongo told a harrowing tale of his family’s experiences in Africa during what are known as the First and Second Congo Wars. He explained that in 1996, the Tutsi Rwandan government funded a rebellion, led by Laurent Kabila, to overthrow Mobuto Sese Seko, the longtime ruler of the Congo (then known as Zaire). The war lasted six months. Because Ms. Makombe was Tutsi, she was in danger; Mr. Kasongo testified that in Kinshasa, Tutsi civilians were burned alive in the streets. He contacted the International Red Cross, which evacuated the family to Brazzaville, across the Congo River in the neighboring Republic of the Congo. Mr. Kasongo then brought the family to Rwanda, whose government had offered asylum to Congolese Tutsi. In

Rwanda, however, Mr. Kasongo experienced difficulties because his physical experience led people to believe he was Hutu.<sup>2</sup> After Kabila prevailed, ending the First Congo War, the family returned to Kinshasa, where Mr. Kasongo opened a grocery store and established businesses in transportation and construction to assist with rebuilding the country's infrastructure. (*Id.*)

In August 1998, the Second Congo War began with a rebellion against Kabila. Mr. Kasongo described a scene in Kinshasa of massacres and generalized killings. One week after the war began, Mr. Kasongo received a phone call at work, informing him that soldiers had come to his house and taken his wife. A maid reported that thirteen or fourteen soldiers had come to the house, and that four had gone into Ms. Makombe's bedroom, beaten her, and raped her. After learning that his wife was being held in a detention camp for Tutsi and Rwandans, Mr. Kasongo persuaded the camp's commanding officer to allow him to take her. Because she was Tutsi, it was not safe to take her to a hospital; his cousin's wife, however, was a medical student who helped care for her. (*Id.*)

The family hid in the house for the next fourteen months. Although Mr. Kasongo approached both the International Red Cross and Catholic Charities, they were unable to help the family escape Kinshasa because the borders were closed. Eventually, with the assistance of the United States government, the family was evacuated to a refugee center and ultimately to a transient camp in Cameroon. There, Ms. Makombe was diagnosed with HIV. According to Mr. Kasongo, she never knew how she had contracted the virus; in fact, he had been advised not to tell his wife that she had been raped during the soldiers' assault, because the knowledge might

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<sup>2</sup> Although Mr. Kasongo did not testify as to the significance of Tutsi and Hutu lineage, the court assumes familiarity with the Rwandan genocide of 1994, in which hundreds of thousands of Tutsi, along with Hutu sympathizers, were slaughtered before Tutsi rebel forces overthrew the Hutu government in July 1994. See *generally* Philip Gourevitch, *WE WISH TO INFORM YOU THAT TOMORROW WE WILL BE KILLED WITH OUR FAMILIES: STORIES FROM RWANDA* (1998).

prove too traumatic.<sup>3</sup> (*Id.*)

In July 2000, the family came to Chicago and moved into a small one-bedroom apartment in the Uptown neighborhood. Ms. Makombe immediately began receiving treatment at the Clinic. (*Id.*)

## **2. The Clinic**

The Clinic, which has been known since 2003 as Heartland Health Outreach, is a wholly owned subsidiary of Heartland Alliance for Human Needs and Human Rights (“Heartland”). (Nelson 12/12/06.) Heartland provides a number of programs and services, including primary medical care, mental health and addiction services, housing and homeless outreach services, and refugee resettlement programs. (*Id.*; see <http://www.heartlandalliance.org/index.html>.) The Clinic operates a community health center for the homeless and working poor, and an HIV/AIDS program that serves approximately 300 un- and under-insured patients. (Nelson 12/12/06; Tornabene 12/11/06.) The Clinic receives approximately two-thirds of its funding from the federal government. (Nelson 12/12/06.)

In 1999, Heartland and the Clinic instituted a program specifically for HIV-positive refugees. (Tornabene 12/11/06.) The Clinic’s Executive Director, Heidi Nelson, explained that in the late 1990s, the Clinton administration created a waiver program as an exception to a federal law that precludes individuals with communicable diseases from entering the United States. The waiver allowed HIV-positive refugees to apply for resettlement in the United States if a healthcare provider and refugee resettlement agency could provide care for them. Because Heartland already provided separate HIV primary care and refugee resettlement services, the Clinic was able to quickly set up the program. According to Nelson, the program has been successful at achieving dramatic medical improvements in HIV-positive refugees, and serves as a model for similar programs throughout the

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<sup>3</sup> Mr. Kasongo does not have HIV; he testified that his wife lost interest in sexual relations after the assault.

country. (Nelson 12/12/06.) In 2001, the program treated approximately 80 HIV-positive refugees and their families.<sup>4</sup> (Tornabene 12/11/06.)

As of October 2001, the Clinic as a whole employed three doctors, five or six nurse practitioners, eight or nine registered nurses, and two midwives. (Tornabene 12/11/06.) At the times relevant to this lawsuit, Nelson was the Executive Director of the Clinic, and Mary Lynn Everson was the managing director; neither is a physician. (Nelson 12/12/06; Everson 12/12/06.) Dr. Katz was the Clinic's medical director, and Tornabene, a licensed advanced practice nurse ("APN"), was the clinical director of primary care services. (Nelson 12/12/06; Tornabene 12/11/06.) Other relevant Clinic personnel include Celine Boers, an APN, (Boers 12/11/06), and Tammy Falk, a registered nurse ("RN"). (Falk 12/12/06.) Dr. Katz, Tornabene, Boers, and Falk all provided at least some degree of care to Ms. Makombe at the Clinic.

Tornabene was the primary healthcare provider<sup>5</sup> for the majority of patients in the HIV-positive refugee program, including Ms. Makombe. (Tornabene 12/11/06.) In her testimony, Tornabene explained that treating HIV-positive refugees presents unique challenges, as many patients come from countries where they received little or no education, and may have endured torture and other trauma. (*Id.*) Falk was the nurse case manager for the HIV-positive refugee patients and their families, working primarily with Tornabene. (*Id.*; Falk 12/12/06.) Falk's role as a nurse and case manager included assisting patients with social service needs, such as housing issues. (*Id.*) She occasionally delivered medications to her patients' homes, or picked up food for them. (*Id.*) Boers had no specific role in the HIV-positive refugee program. (Nelson 12/12/06.)

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<sup>4</sup> Although testimony at trial did not explicitly establish that Ms. Makombe's family came to the United States via the waiver program and/or with Heartland's assistance, the court notes that immigration documents indicate that Ms. Makombe indeed applied for a waiver, (000122), and infers, from the fact that she began receiving treatment at the Clinic immediately upon her arrival, that Heartland was involved.

<sup>5</sup> According to Nelson, a physician or an APN is a "provider," but an RN is not. The court adopts this terminology for purposes of this opinion.

Tornabene practiced pursuant to a collaborative agreement (“CA”) with Dr. Katz, the scope of which is somewhat unclear. The CA states that an APN must obtain a consultation when presented with a clinical situation beyond his or her scope of practice, expertise or experience; and that in the event of disagreement between the APN and the collaborating physician regarding a patient’s diagnosis or treatment, the physician’s decision prevails. (Heartland Health Outreach Advanced Practice Nurse Collaborative Agreement, Pl.’s Ex. 8; Tornabene 12/11/06.) Tornabene testified that prior to Ms. Makombe’s admission to Weiss on October 22, 2001, shortly before her death, nothing in her care or treatment was beyond the scope of Tornabene’s expertise as a nurse practitioner. (Tornabene 12/11/06.) Dr. Katz testified that she and Tornabene “worked as equals,” that they consulted with each other as needed, and that physician/nurse practitioner distinctions were “pretty much lost” in practice. (Katz 12/13/06.) Dr. Katz also noted that Tornabene had more experience working with HIV patients than she did. (*Id.*)

Tornabene and Dr. Katz gave somewhat inconsistent answers, however, when asked who was “ultimately responsible” for Ms. Makombe’s care and treatment at the Clinic under the CA. Tornabene testified that she was responsible for Ms. Makombe’s care; yet she acknowledged testifying in her deposition that Dr. Katz was “ultimately responsible for the care and treatment of Jacqueline Makombe with regard to doctors.” (Tornabene 12/11/06.) When asked in her deposition if she was ultimately responsible for Ms. Makombe’s care and treatment, Dr. Katz answered, “I don’t believe so”; and when asked that question at trial, replied that Ms. Makombe “was not [her] patient,” and that Tornabene was “primarily responsible.” (Katz 12/13/06.) Dr. Katz further testified that she had no oversight obligations pursuant to the CA, was unclear as to the responsibility of a collaborating physician in 2001, and did not know who was ultimately responsible for Ms. Makombe’s care. (*Id.*) Plaintiff’s expert, Dr. Rumans, testified that under the CA, Dr. Katz had the “ultimate responsibility” for Ms. Makombe’s care, and was “primarily responsible.” (Rumans 12/14/06.)

Clinic Executive Director Nelson testified that Dr. Katz was primarily responsible for promulgating policies and procedures pertaining to care and treatment at the Clinic, and for ensuring that clinical guidelines relating to patient care were known by the medical staff. (Nelson 12/12/06.) Tornabene, as the clinical director of primary care services, was responsible for the “maintenance” of policies and procedures for primary care services in 2001, and for supervising the other APNs and RNs. (*Id.*) Nelson explained that nurse practitioners such as Tornabene are important to an outpatient medical clinic because they can perform the same tasks as physicians, can spend more time with patients and perhaps relate better to them, but command lesser salaries and are thus cost effective. (*Id.*)

Dr. Rumans and Bradford Farrington, an APN who testified for the government, each asserted that the standard of care is identical for a physician and a nurse practitioner. (Rumans 12/14/06; Farrington 12/15/06.) Neither party has argued to the contrary.

## **B. HIV/AIDS, Zerit, and Lactic Acidosis**

### **1. HIV/AIDS and Zerit**

Acquired immunodeficiency syndrome, or AIDS, results from infection with the human immunodeficiency virus, or HIV. See DORLAND’S ILLUSTRATED MEDICAL DICTIONARY 1623 (28th ed. 1994) (“DORLAND’S”). An HIV-positive patient is diagnosed with AIDS upon the presence of either (1) an AIDS-defining illness, as defined by the Centers for Disease Control (“CDC”); or a T-cell count below 200.<sup>6</sup> (Tornabene 12/11/06.) According to Tornabene, three classes of drugs were used in 2001 to treat AIDS: nucleoside reverse transcriptase inhibitors (“NRTIs”); non-nucleoside reverse transcriptase inhibitors; and protease inhibitors. (*Id.*) A common treatment is known as Highly Active Antiretroviral Therapy, or HAART, which consists of a combination of antiretroviral

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<sup>6</sup> Tornabene explained that T-cells are the chief functioning cells of the immune system; the more depleted the level of such cells, the more likely an individual will become ill. (Tornabene 12/11/06.) At trial, the term “T cell” was used interchangeably with the term “CD4.”

drugs from the different classes. See Treatment of HIV Infection, <http://www.niaid.nih.gov/factsheets/treat-hiv.htm>.

Plaintiff's expert, Dr. Rumans, explained that HIV uses an enzyme, reverse transcriptase, to replicate itself by utilizing the DNA machinery in human cells. (Rumans 12/14/06.) NRTIs block that replication process by inserting themselves into the DNA replication chain, thereby preventing the virus from reproducing. (*Id.*) In the early 1980s, the NRTI drug AZT was the only treatment available for AIDS. (Rumans 12/14/06.) Zerit, or stavudine, is a newer NRTI that became available in 1994. (*Id.*) According to Plaintiff's expert Dr. Novak, who testified as to Ms. Makombe's life expectancy, HAART treatment that includes NRTIs such as Zerit has transformed HIV from a "death sentence" into a chronic disease, similar to diabetes, that "can be managed for an indefinite period of time." (Novak 12/14/06.)

## **2. Lactic Acidosis**

### **a. As Side Effect of Zerit**

A rare but often lethal side effect of NRTIs, including Zerit and AZT, is a condition known as lactic acidosis. Lactic acidosis is "a metabolic acidosis occurring as a result of excess lactic acid in the blood, due to conditions causing impaired cellular respiration." DORLAND'S, at 16. Dr. Rumans explained that lactic acidosis can occur when the energy process within human cells, a process known as the "Krebs cycle," shifts from an aerobic to an anaerobic metabolism. (Rumans 12/14/06.) Ordinarily, mitochondria within cells metabolize glucose into energy; lactic acidosis results when the mitochondria are unable to take up the glucose, which instead turns into lactic acid (lactate). (*Id.*; Kessler 12/19/06.) NRTIs such as Zerit can cause lactic acidosis through mitochondrial toxicity, which occurs when the NRTIs bind themselves to cellular enzymes and shut down energy production within cells. (Rumans 12/14/06.) As a result of this mitochondrial dysfunction, the body is unable to eliminate the excess lactate, which continues to build up in the bloodstream. (Kessler 12/19/06.) Elevated levels of lactic acid, referred to as hyperlactatemia, can

lead to severe lactic acidosis if the catalyst for the elevated lactate, e.g., the Zerit, is allowed to continue.<sup>7</sup> (Rumans 12/14/06.) Patients who die from lactic acidosis typically do so as a result of respiratory failure. (*Id.*)

**b. Signs and Symptoms**

The parties do not dispute the nature of the signs and symptoms of HAART-related lactic acidosis. Clinical guidelines published by the DHHS and available on the CDC's website in August 2001, titled "Guidelines for the Use of Antiretroviral Agents in HIV-Infected Adults and Adolescents" (the "CDC Guidelines"), describe those signs and symptoms.<sup>8</sup> (CDC Guidelines, Pl.'s Ex. 8, at 19.) According to the CDC Guidelines, the initial clinical presentation may include "nonspecific gastrointestinal symptoms" and dyspnea. (*Id.*) Dyspnea, both parties' experts testified, means shortness of breath, or the patient's perception or sensation thereof. (Rumans 12/14/01; Kessler 12/19/01.) Other symptoms "may include otherwise unexplained onset and persistence of abdominal distention, nausea, abdominal pain, vomiting, diarrhea, anorexia, generalized weakness, weight loss, and hepatomegaly" (enlarged liver). (CDC Guidelines, at 19).

Dr. Rumans provided a more detailed, and unchallenged, explanation of the progression of lactic acidosis. The initial symptoms are gastrointestinal: nausea, possibly vomiting, loss of appetite, and abdominal discomfort ranging from pain to "just an unpleasant feeling of fullness and distension." (Rumans 12/14/06.) Weight loss may occur. (*Id.*) Next, the patient may complain of

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<sup>7</sup> The lactic acidosis at issue in this case is the type of lactic acidosis related to NRTIs such as Zerit. Lactic acidosis occurs in other situations as well, such as in hospital settings where a patient has experienced tissue trauma or has sepsis. (Tornabene 12/11/06.)

<sup>8</sup> The CDC Guidelines entered into evidence at trial are dated August 13, 2001, which date Plaintiff emphasizes as falling at the beginning of the time frame in which Ms. Makombe began showing signs and symptoms of lactic acidosis, as discussed below. The court notes, however, that identical sections discussing HAART-associated lactic acidosis appear in archived versions of CDC guidelines, published on February 5, 2001 and April 23, 2001, that are available for download on a DHHS website. See Archived Guidelines, <http://www.hivatis.org/Guidelines/ArchivedGuidelines.aspx>.

inadequate respiration. (*Id.*) Dr. Rumans explained that lactic acidosis effects an alteration in the respiratory system, but one that occurs slowly; thus, respiratory problems may not be “readily identifiable” to someone looking at the patient, but the patient nonetheless complains of feeling breathless. (*Id.*) As the condition progresses, the patient complains of fatigue and weakness, and begins to develop muscle aches and pains, or “myalgia,” due to excess lactate in the bloodstream. (*Id.*) Paresthesia, or numbness and tingling in the extremities, may develop and may mimic or become a type of peripheral neuropathy (numbness and pain resulting from nerve damage). (*Id.*)

Both parties’ experts agreed that despite the possibility of lactic acidosis, routine monitoring of lactate levels in patients taking NRTIs, through serum lactate blood testing, is unnecessary because these patients can have elevated lactate levels without showing signs of, or developing, acidosis. (Rumans 12/14/06; Kessler 12/19/06.) A serum lactate test is required once a patient shows signs and symptoms sufficient to place lactic acidosis on a differential diagnosis;<sup>9</sup> if the test shows elevated lactate levels, then an arterial blood gas test can confirm the presence of the condition. (Rumans 12/14/06; Tornabene 12/11/06.) It is further undisputed that the treatment for lactic acidosis caused by Zerit is the immediate cessation of the use of Zerit. (Rumans 12/14/06; Katz 12/13/06.)

**c. Frequency**

The parties agree that lactic acidosis is a rare side effect of Zerit; the government characterizes it as exceedingly so. Tornabene testified that lactic acidosis is a “very rare side effect” of antiretroviral drugs, and is “very uncommon”; she estimated its frequency at “somewhere around 3.2 cases per 1000 person years.” (Tornabene 12/11/06.) Dr. Katz referred to lactic acidosis as “incredibly rare” in 2001. (Katz 12/13/06.) Dr. Kessler called it a “very rare, rare

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<sup>9</sup> As a general matter, a “differential diagnosis” is “the determination of which one of two or more diseases or conditions a patient is suffering from, by systematically comparing and contrasting their clinical findings.” DORLAND’S, at 458. As will be discussed, the witnesses in this case offered slightly different interpretations of the term in the context of lactic acidosis.

disease,” and testified that of 681,000 prescriptions for Zerit issued in 1997, only thirty-nine cases of lactic acidosis were reported to the FDA. (Kessler 12/19/06.) He acknowledged, however, that there may have been other cases that were not reported to the FDA. (*Id.*)

Plaintiff, while not denying that lactic acidosis is rare, presented uncontradicted evidence that it was nonetheless a well-known side effect of Zerit by 2001 and probably for several years before that. Defendant’s expert, Dr. Kessler, acknowledged that there had been a “spike” in Zerit-related lactic acidosis in 1997, and that an article published by the FDA in 1999 had highlighted lactic acidosis as a side effect of Zerit. (*Id.*) Dr. Rumans testified that lactic acidosis had been linked to the NRTI drug AZT as far back as 1989, and that by the time Zerit appeared in 1994, lactic acidosis was a known side effect of NRTIs. (Rumans 12/14/06.) As early as 1996, according to Dr. Rumans, there had been reports of a link between lactic acidosis and Zerit specifically, and by 2000 there were warnings in the medical literature and at HIV care provider meetings. (*Id.*) By 2001, Dr. Rumans testified, the linkage between Zerit and lactic acidosis had thus been “clearly previously established” as far back as 1997 within the community of AIDS and HIV treatment providers. (*Id.*)

As noted, the CDC Guidelines specifically warned of HAART-related lactic acidosis in 2001. In addition, the FDA-approved package insert for Zerit in 1999, as replicated in the Physicians’ Desk Reference (“PDR”), contained a “black box” warning of reported cases of fatal lactic acidosis with the use of NRTIs including Zerit.<sup>10</sup> (1999 PDR, PI.’s Ex. 52; Kessler 12/19/06.) The same black box warning appeared in package inserts in 2000 and 2001. (2000 PDR, PI.’s Ex. 51; 2001 PDR, PI.’s Ex. 50.) The 1999 and 2000 versions, in a separate “Warnings” section, added that the majority of reports involved women, and the 2001 version elaborated that obesity and prolonged

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<sup>10</sup> The 1999 black box warning, appearing at the beginning of the PDR section for Zerit, specifically states: “Warning: lactic acidosis and severe hepatomegaly with steatosis, including fatal cases, have been reported with the use of antiretroviral nucleoside analogues alone or in combination, including stavudine.” (1999 PDR, PI.’s Ex. 52.)

NRTI exposure were additional risk factors. (2001 PDR, Pl.'s Ex. 50.) Plaintiff additionally points to an article in the Journal of the Association of Nurses in AIDS Care, published in the fall of 2000, that specifically addressed lactic acidosis as a side effect of NRTI medications. (Patricia M. Caffrey, *Lactic Acidosis Associated With Nucleoside Reverse Transcriptase Inhibitors*, 11 J. ASSOC. NURSES AIDS CARE 91 (2000), Pl.'s Ex. 43.) The article, written by an RN, discusses early nursing intervention, including early identification and symptom management of lactic acidosis. (*Id.*)

**C. Ms. Makombe's Treatment at the Clinic**

**1. Treatment between July 2000 and August 10, 2001**

Ms. Makombe first came to the Clinic on July 24, 2000, immediately after her arrival in the United States, at which time her condition was evaluated and a nurse took her personal and medical history. (Tornabene 12/11/06; 000004–08, 000017-19.) Although she had no AIDS-defining illness, her T-cell (CD4) count was 48, indicating a diagnosis of AIDS, and her viral load was more than 90,000.<sup>11</sup> (Tornabene 12/11/06; 000010.) Mary Tornabene, who testified that she was Ms. Makombe's primary healthcare provider throughout her treatment, first saw her on August 15, 2000. (Tornabene 12/11/06.) Tornabene's August 15 treatment notes state that Ms. Makombe felt "sad" and "depressed," but that she denied the "usual problems" associated with AIDS, such as sores in the mouth or chest pain. (000013.) Tornabene prescribed Zithromax and Bactrim as prophylactic antibiotics to protect against pneumonia and other infections. (Falk 12/12/06; 000019.)

At her next visit to the Clinic on September 19, 2000, Ms. Makombe reported no side effects from the medications. (000019.) She began HAART treatment with a combination of AZT and

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<sup>11</sup> Viral load measures the amount of HIV an individual has within a specific volume. (Rumans 12/14/06.) Tornabene testified that Ms. Makombe's initial T-cell count of 48 was "very low," and her viral load of more than 90,000 was "very high." (Tornabene 12/11/06.)

Although Ms. Makombe's medical history included malaria and typhoid, Dr. Rumans testified that neither are considered opportunistic AIDS-related infections. (Rumans 12/14/06.)

Sustiva.<sup>12</sup> (Falk 12/12/06; 000021.) On September 26, 2000, she complained of a “slightly queasy stomach” after taking the Bactrim, but reported that the feeling went away when she drank a glass of water. (000021-22.) On October 24, 2000, Tornabene noted “side effects from [the] Zithromax” including nausea; Tornabene discontinued that medication, and gave instructions to take the Bactrim with food and a large glass of water. (000022-23.) After routine blood tests revealed that the AZT was causing a low neutrophil count,<sup>13</sup> Tornabene on October 26, 2000 switched Ms. Makombe from AZT to Zerit. (000023-24; Tornabene 12/11/06.) From that point until April 2001, Ms. Makombe presented with no serious problems; nurse Falk testified that she reported only “run of the mill complaints.” (Falk 12/12/06.) Ms. Makombe responded well to the Zerit. By January 23, 2001, her T-cell count was up to 112, and her viral load was less than 50, or undetectable. (000009.)

On April 17, 2001, Ms. Makombe reported dizziness and blackouts when she had “a lot of things to do at one time” or when she felt “overwhelmed.” (000028-29.) Through a French interpreter,<sup>14</sup> she described having been beaten unconscious during wartime in the Congo. (*Id.*) In her subjective assessment, Tornabene wrote “Prayer is treatment of choice for this person.” (000029.) Tornabene made an assessment of post-traumatic stress disorder (“PTSD”) and offered

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<sup>12</sup> AZT is prescribed under the name Combivir; Sustiva is a non-nucleoside reverse transcriptase inhibitor, often used in combination with NRTIs in HAART treatment. See Treatment of HIV Infection, <http://www.niaid.nih.gov/factsheets/treat-hiv.htm>.

<sup>13</sup> Dr. Rumans explained that neutrophils are components of white blood cells that are important in responding to different types of bacterial infections. (Rumans 12/14/06.)

<sup>14</sup> Tornabene testified that Ms. Makombe required an interpreter early on during her care at the Clinic, but that she was taking English classes and was increasingly able to communicate on her own. (Tornabene 12/11/06.) Falk testified that Ms. Makombe’s English was “pretty good,” and that while some patients require an interpreter for proper care, Ms. Makombe was not one of them. (Falk 12/12/06.) She further testified that there was no instance in which Ms. Makombe’s complaints were made more clear by the presence of an interpreter. (*Id.*)

a mental health referral, which Ms. Makombe refused.<sup>15</sup> (000029.)

Also on April 17, Tornabene completed a “Report of Incapacity” for the Illinois Department of Human Services. (000164-000167.) Tornabene listed AIDS and PTSD as the “chief complaints of patient.” (000164.) In the section concerning limitations on activities, she indicated that Ms. Makombe had a 20-50% reduced capacity in the activities of walking, bending, turning, standing, climbing and stooping; reduced capacity of up to 20% in sitting, speaking, pushing, travel, and grasping manipulations; and a lifting limit of ten pounds. (000167.) Where the form called for observations of mental status, Tornabene made a notation that Ms. Makombe suffered from “severe PTSD” and depression, and further wrote: “needs intensive therapy—refusing at present.” (*Id.*) These mental impairments resulted, in Tornabene’s estimation, in a reduced capacity of more than 50% in activities of daily living, in social functioning, and in concentration, persistence, and pace. (*Id.*)

On May 14, 2001, nurse Falk went to Ms. Makombe’s home after receiving a phone call in which the caller, whom Falk does not recall, reported that Ms. Makombe had experienced dizzy spells and had fallen. (Falk 12/12/06.) Falk testified that she made the home visit out of concern for Ms. Makombe, and that she found Ms. Makombe sleepy yet easily aroused. (*Id.*) Her written assessment contains the notation “PTSD?”. (000035.) Ms. Makombe came to the Clinic the following day, and saw both Falk and Tornabene. (Tornabene 12/11/06.) Tornabene noted that Ms. Makombe complained that she “gets to feeling ‘too much’ and then gets dizzy and passes out.” (*Id.*; 000035.) Tornabene assessed her symptoms as “probably PTSD,” and referred Ms. Makombe to two Heartland programs for refugees that have experienced torture or other trauma, the Kovler

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<sup>15</sup> The government attempted to elicit testimony from nurse Falk that the diagnosis of PTSD in Ms. Makombe’s records came from an (unidentified) psychiatrist rather than Tornabene. (Falk 12/12/06.) The court sustained Plaintiff’s objection on relevance grounds.

Center<sup>16</sup> and Refugee Mental Health. (*Id.*; 000036.) Tornabene testified that she wanted Ms. Makombe to be evaluated by a psychiatrist at Kovler, but that Ms. Makombe never was evaluated.<sup>17</sup> (Tornabene 12/11/06.)

Ms. Makombe returned to the Clinic on May 29, 2001 for a refill of her medications, seeing only nurse Falk, and reporting no complaints. (Falk 12/12/06.) On July 17, 2001, again seeing only Falk, she reported, through an interpreter, having fainted while doing dishes, and that Mr. Kasongo had had difficulty reviving her for several hours afterward. (*Id.*; 000036.) Falk scheduled an EEG for July 19, the results of which showed a possible mild HIV-related encephalopathy, which according to Tornabene can cause various neurological problems, including dementia. (Tornabene 12/11/06.) Tornabene scheduled an MRI, but Ms. Makombe was unable to complete it; she could not tolerate the test due to its closeness and sounds that resemble gunfire.<sup>18</sup> (*Id.*)

Plaintiff does not assert that any of Ms. Makombe's above complaints or presenting symptoms, prior to August 10, 2001, constituted signs and symptoms of lactic acidosis.

## **2. Clinic Visits on August 10 and August 28, 2001**

On August 10, 2001, Ms. Makombe presented at the Clinic complaining of nausea, gastrointestinal ("GI") discomfort, and feelings of fullness and tenderness in both breasts. (000039.) She saw only nurse Falk, whom she told that she wanted no more tests, and that her symptoms

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<sup>16</sup> The Marjorie Kovler Center for the Treatment of Survivors of Torture "provides comprehensive services to survivors of officially sanctioned government or political torture who live in Chicago and the Midwest." <http://www.heartlandalliance.org/kovler/>.

<sup>17</sup> Tornabene did not testify as to whether Ms. Makombe received treatment from Refugee Mental Health. The court notes, however, that Ms. Makombe appeared at several Clinic appointments with an interpreter named Martine, whom nurse Falk in her treatment notes described as Ms. Makombe's "mental health case worker from [Heartland] Refugee Mental Health." (000039.)

<sup>18</sup> When Falk attempted to reschedule the MRI, Ms. Makombe asserted that she "would rather die than do the test again." (000040.) The failed MRI does not appear relevant to this case, as the government does not contend that any test refused by Ms. Makombe may have led to an earlier diagnosis of lactic acidosis.

were “God’s way of telling me not to take meds anymore.” (*Id.*) Ms. Makombe denied missing any doses of her medication, however. (*Id.*) Falk did a pregnancy test, which was negative, and offered over-the-counter antacids for the “GI discomfort.” (*Id.*) Falk did not consult with Tornabene or another provider on August 10. (Falk 12/12/06.)

Ms. Makombe returned to the Clinic for another scheduled appointment on August 28, 2001, seeing both Falk and Tornabene. (Tornabene 12/11/06; 000041.) She complained of “continued GI distress,” especially when eating spicy food, but reported that the antacids helped. (*Id.*) She reported a continued feeling of fullness in her breasts. (000041.) Tornabene’s notes indicate that Ms. Makombe reported “100% adherence to HAART” and denied missing any doses. (*Id.*)

Tornabene ordered pituitary gland tests to see if a pituitary discovery could be the source of Ms. Makombe’s breast enlargement and weight gain. (Tornabene 12/11/06; Falk 12/12/06.) According to her “HIV Flowsheet,” Ms. Makombe weighed 153 lbs. in September 2000; she weighed 179 lbs. in March 2001, and 194 lbs. on August 28, 2001. (000009; Falk 12/12/06.) Tornabene testified that the weight gain was “a good thing.” (Tornabene 12/11/06.) Ms. Makombe had not been weighed at the Clinic between March and August, and after the pituitary tests results came back normal, no follow-up was done with regard to her weight. (Falk 12/12/06.)

Ms. Makombe also came to the Clinic on September 7, 2001 for a tetanus shot. (*Id.*) She told nurse Falk at that time that her breast swelling and tenderness had decreased. (*Id.*; 000043.)

### **3. September 18 and September 26, 2001**

On September 18, 2001, Ms. Makombe returned to the Clinic for a scheduled appointment, seeing both Falk and Tornabene. (000042, 000044.) She reported doing “generally well,” and that she no longer had headaches or blackouts. (000042; Tornabene 12/11/06.) She also reported, however, a “scant amount” of yellow bilious emesis (vomit), as well as abdominal pain in the upper left quadrant. (000042.) Tornabene found Ms. Makombe’s abdomen large and firm. (Tornabene 12/11/06.) An X-ray showed a normal abdomen. (*Id.*) To rule out gallbladder disease, Tornabene

scheduled an ultrasound of the gallbladder, spleen, liver, and pancreas. (*Id.*; 000044.)

On September 26, 2001, Ms. Makombe arrived at the Clinic without an appointment and was triaged by nurse Falk. (000046; Tornabene 12/11/06.) Ms. Makombe complained of throat pain while swallowing, and reported that she had vomited three times the night before. (000046.) Falk's assessment on the triage form<sup>19</sup> indicated "probable viral flu (Indigestion)," and Falk recommended rest, clear liquids, a bland diet, and antacids. (*Id.*) Neither Tornabene nor any other provider examined Ms. Makombe on September 26. (Tornabene 12/11/06.)

#### **4. October 1, 2001**

Ms. Makombe again came to the Clinic without an appointment on October 1, 2001, and was again triaged by nurse Falk. (000047; Tornabene 12/11/06.) No interpreter was present. (000047.) The triage form notes a "continued" complaint of "no eating," abdominal pain in the mid epigastric area (below the breast bone), vomiting, and constipation. (*Id.*) Falk also wrote that Ms. Makombe "states 'respirations are low.'" (*Id.*) According to Falk, Ms. Makombe made this comment in "not terribly broken" English. (Falk 12/12/06.)

Falk testified that she took vital signs: temperature, blood pressure, pulse, and respiratory rate. (*Id.*) The triage form notes a respiratory rate of 22, which Tornabene testified is normal. (000047; Tornabene 12/11/06.) Falk found no bowel obstruction that could have been causing abdominal pain. (000047; Falk 12/12/06.) Falk indicated "NAD", or "no apparent distress," on the triage form. (000047.) The box labeled "shortness of breath" is unchecked on the form, (*id.*), and no pulse oximeter test (finger test) was done to measure the level of oxygenation in Ms. Makombe's blood.<sup>20</sup> (Tornabene 12/11/06.)

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<sup>19</sup> When a patient came to the Clinic without an appointment, a triage form was completed, instead of SOAP (subjective/objective/assessment/plan) notes. (Tornabene 12/11/06.)

<sup>20</sup> Although no pulse oximeter (SP O2) test was done on October 1, the testimony at trial did not establish that such a test is called for in any instance in which a patient complains of  
(continued...)

Although Ms. Makombe was Tornabene's patient, Tornabene was not contacted on October 1; instead, Falk consulted with Celine Boers, another APN.<sup>21</sup> (*Id.*; Falk 12/12/06.) Boers diagnosed constipation and gave instructions for treatment with Milk of Magnesia. (Boers 12/11/06.) Beyond that diagnosis, the extent of Falk's consultation with Boers is unclear. It is undisputed that Boers did not examine Ms. Makombe on October 1; Boers further testified that she has no recollection even of talking to Falk on October 1. (*Id.*) Falk testified that she told Boers about the constipation, but that she also showed Boers the triage form, which noted other symptoms, including vomiting. (Falk 12/12/06.) Boers does not remember the conversation, nor ever seeing the triage form, and testified that she would not be likely to diagnose constipation, or to prescribe Milk of Magnesia, for a patient who was vomiting. (Boers 12/11/06.)

Boers also ordered liver function tests ("LFTs"). (000047.) Although neither Boers nor Falk testified as to why LFTs were ordered, Falk made the notation "?Lft's" in her nursing assessment on the triage form, (*id.*), suggesting that perhaps Falk proposed the tests to Boers, who agreed.

Falk's consultation with Boers on October 1 amounted to Boers' only involvement in the care and treatment of Ms. Makombe. (Boers 12/11/06.)

## **5. October 3, 2001**

Ms. Makombe made a third unscheduled visit to the Clinic on October 3, 2001. (000048.) Her caseworker Martine was present to interpret. (*Id.*) Falk's triage form notes complaints of vomiting, decreased appetite, weakness, dizziness, shortness of breath, and increased breast size and tenderness. (*Id.*) Falk again took vital signs; Ms. Makombe's respiratory rate was 20, which

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<sup>20</sup>(...continued)

shortness of breath. Dr. Kessler, the government's medical expert, testified that because Ms. Makombe's respiratory rate was normal and she was not in distress, no SP O2 test was required to pursue Ms. Makombe's complaint of low respirations. (Kessler 12/19/06.) The court does not understand Plaintiff to argue that such a test was required on October 1; indeed, as discussed below, the expert testimony indicated that an SP O2 test is unhelpful in diagnosing lactic acidosis.

<sup>21</sup> Tornabene worked part-time at the Clinic, two days a week. (Tornabene 12/12/06.)

was still normal. (*Id.*; Tornabene 12/11/06.) Falk also noted on the form that she had called for the results of the ultrasound scan that had been conducted on October 1; that test showed that Ms. Makombe had gallstones and that her liver was normal. (000048; 000092; Tornabene 12/11/06.) As her “nursing assessment,” Falk recorded that Ms. Makombe suffered from gallstones, “possible gastric inflammation,” and depression, though the latter observation was preceded by a question mark. (000048.) Falk again indicated “NAD” on the triage form. (*Id.*) She also recommended that Ms. Makombe see a nutritionist for help with the decreased appetite, and made a referral for home help. (*Id.*; Falk 12/12/06.) Ms. Makombe refused the home help referral, telling Falk that her husband “might get mad.” (Falk 12/12/06.)

Falk consulted with Dr. Katz on October 3; Tornabene again was not contacted. (Falk 12/12/06; Tornabene 12/11/06.) Falk testified that she described all of Ms. Makombe’s signs and symptoms in her conversation with Dr. Katz. (Falk 12/12/06.) Dr. Katz herself did not examine Ms. Makombe, and did not review Ms. Makombe’s medical records. (Katz 12/13/06.) Dr. Katz prescribed Ranitidine, a drug used to treat gastroesophageal reflux disease (“GERD”),<sup>22</sup> and instructed Falk to schedule an appointment for Ms. Makombe with a surgeon for a gallbladder evaluation. (*Id.*; 000048.) Falk testified that Dr. Katz also instructed her to do a pulse oximeter test; the test showed an oxygenation rate (SP O<sub>2</sub>) of 98%, which is normal. (Falk 12/12/06; Tornabene 12/11/06.)

#### **6. October 9, 2001**

On October 9, 2001, Ms. Makombe came to the Clinic for a scheduled appointment. (Tornabene 12/11/06.) No interpreter was present. (*Id.*) She saw both Tornabene and Falk. (*Id.*) To Falk, she complained of pain deep inside both legs from the knees to the soles of her feet, and reported that the skin on both legs felt numb. (000049.) She told Falk that her breasts were

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<sup>22</sup> The brand name for Ranitidine is Zantac, which is now a common over-the-counter medication for reducing stomach acid. See <http://www.drugs.com/cdi/ranitidine.html>.

“bothering [her] very much.” (*Id.*) Falk wrote that Ms. Makombe “still feels that breathing is very low.” (*Id.*) Ms. Makombe also reported that her appetite and eating had improved; Falk noted, however, that Ms. Makombe had lost “10 lbs.” since August.<sup>23</sup> (*Id.*) Falk measured a respiratory rate of 20; neither Falk nor Tornabene did a pulse oximeter test. (*Id.*; Tornabene 12/11/06.)

Tornabene noted “multiple somatic<sup>24</sup> [complaints] including bilateral leg soreness” with “some paresthesias,” and that Ms. Makombe “feels breathing so heavy because breasts are so big.” (000050.) Tornabene then conducted a physical exam, which she described in her testimony as “almost a complete exam.” (Tornabene 12/11/06.) She found Ms. Makombe’s lungs clear, her mouth fine, and her abdomen soft, not tender. (*Id.*) Tornabene recalled that Ms. Makombe “looked great on that day. She looked really really good.” (*Id.*) Tornabene assessed AIDS, PTSD, and gallstones, and told Ms. Makombe to return in one to two weeks because she wanted to keep a “close eye” on Ms. Makombe’s complaints. (*Id.*)

Tornabene testified, and her treatment notes indicate, that she considered stopping the Zerit treatment on October 9. (*Id.*; 000050.) This was not out of concern about lactic acidosis, however. Tornabene testified that paresthesia is a common side effect of Zerit, and that there had been (unidentified) warnings by 2001 of peripheral neuropathy, a symptom that might be alleviated by removal of Zerit. (Tornabene 12/11/06.) Dr. Rumans similarly testified that paresthesia caused by a neuropathy was a well-recognized side effect of Zerit in 2001, (Rumans 12/14/01), and the 1999, 2000, and 2001 package inserts for Zerit warned of peripheral neuropathy. (PDRs, Pl.’s Ex. 50-52.) Tornabene testified that she considered stopping the Zerit because it might be causing a neuropathy, and that she discussed this with Ms. Makombe. (Tornabene 12/11/06.) According to

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<sup>23</sup> In fact, Ms. Makombe’s “HIV Flowsheet” shows that she weighed 182 pounds on October 9, signifying a twelve-pound loss from August 28. (000009.)

<sup>24</sup> “Somatic” means pertaining to or characteristic of the “soma” or body. DORLAND’S, at 1544. According to Falk, a somatic complaint is one that a patient perceives or feels, as opposed to a psychosomatic complaint which is imagined. (Falk 12/12/06.)

Tornabene, Ms. Makombe was unwilling to stop taking the Zerit, and instead told Tornabene that she had a friend with tuberculosis who took vitamin B6 for a neuropathy. (*Id.*) Tornabene agreed that taking B6 would do no harm, and that Ms. Makombe could continue the Zerit treatment because Zerit had been effective at keeping her viral load down and her T-cell count stable. (*Id.*)

Tornabene further testified that as of October 9, she was aware of the results of the LFTs that Boers had ordered on October 1. (*Id.*) She did not explain when she reviewed those results, and there is no mention of them in her October 9 notes. (000050.) Those results, which measured liver enzyme levels, indicate an ALT (alanine aminotransferase) of 45 and an AST (aspartate aminotransferase) of 53; normal range for both is between zero and 40. (Tornabene 12/11/06; 000090.) In her testimony, Tornabene nevertheless characterized these results as “essentially normal” because elevated AST and ALT levels are common with HIV. (Tornabene 12/11/06.) In Tornabene’s view, only a reading as high as 80 would be cause for concern. (*Id.*) She acknowledged, however, that patients with lactic acidosis can have elevated liver enzymes. (*Id.*)

On October 15, 2001, Ms. Makombe saw Dr. Gerald Kaplan for the gallstone evaluation that Dr. Katz had ordered on October 3. (000090.) Dr. Kaplan recommended an upper GI endoscopy to rule out GERD. (*Id.*) The record from this visit appears to consist entirely of the referral form, on which Dr. Kaplan’s brief remarks note no complaints other than the GI complaints that were the basis for the referral. (000090.)

#### **7. October 22, 2001**

Ms. Makombe came to the Clinic at 10:30 a.m. on October 22, 2001 without an appointment, complaining of shortness of breath that “feels like suffocating,” severe pain in her ankles and the soles of her feet, and “burning” epigastric pain. (000051.) She reported that she had not been eating or sleeping. (*Id.*) Falk’s notes on the triage form also include vomiting, dizziness, weakness, and increased abdomen size. (*Id.*) October 22 was a Monday; Ms. Makombe told Falk that she had been feeling poorly all weekend, but that the Clinic had been closed. (Falk 12/12/06.) Despite

Ms. Makombe's complaints, Falk did not believe she was in distress because her vital signs were normal.<sup>25</sup> (*Id.*)

After the initial triage, Falk spoke with Tornabene, who did not examine Ms. Makombe. (*Id.*) Falk testified that Tornabene could see Ms. Makombe in an examination room from where Tornabene was sitting at the nurses' station. (*Id.*) Tornabene testified that she saw Ms. Makombe from several feet away and that she "looked awful." (Tornabene 12/11/06.) Tornabene told Falk to send Ms. Makombe to the outpatient lab at Weiss for a STAT<sup>26</sup> serum lactate test and for STAT LFTs, and to stop the HAART medications. (*Id.*; 000051-000052.) She also told Falk to do a pulse oximeter test, which again showed an SP O2 level of 98%. (Falk 12/12/06; 000051.) By this point, lactic acidosis was on the differential diagnosis, (Tornabene 12/11/06); Tornabene's notes from October 22 state that her assessment was to rule out lactic acidosis. (000052.)

According to Tornabene, she wanted Ms. Makombe to go the outpatient lab at Weiss, rather than to the emergency room ("ER"), because the test results would come back more quickly. (*Id.*) Falk testified that when she told Ms. Makombe to go to the outpatient lab, which was several blocks away, Ms. Makombe replied that she would rather go to the ER, asking, "do you want me to die in the street?"<sup>27</sup> (Falk 12/12/06.) Falk testified that because Ms. Makombe was able to walk, she did not call an ambulance. (*Id.*) Ms. Makombe's caseworker Martine accompanied her to Weiss. (*Id.*)

The Weiss lab collected blood at 12:10 p.m. on October 22. (000093.) The results, indicating highly elevated lactate levels, were faxed to the clinic at 2:17 p.m. (Tornabene 12/11/06.)

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<sup>25</sup> The triage form indicates that Ms. Makombe's temperature was 97.6, her blood pressure was 98/64, her pulse was 104, and her respiratory rate was 22. (000051.) Unlike the October 1 and October 3 triage forms completed by Falk, there is no explicit "NAD" indication on the October 22 triage form. (000051.)

<sup>26</sup> "Stat.," an abbreviation for the Latin term "*statim*," means "immediately." DORLAND'S, at 1574.

<sup>27</sup> Although Ms. Makombe's case worker and interpreter Martine was present at some point on October 22, it is unclear whether this comment was made in English or through Martine.

An arterial blood gas test was done at Weiss at 2:16 p.m. (000094.) Ms. Makombe was seen at the ER at Weiss at 4:10 p.m.; ER intake records state that Dr. Katz referred her there. (000157.) The ER had the results of the serum lactate and arterial blood gas tests. (Tornabene 12/11/06.) The ER records indicate that Ms. Makombe's oxygenation rate was 100%, that she was ambulatory, and that her condition was "nonurgent." (000157.) She was subsequently admitted to the hospital. (Tornabene 12/11/06.)

Mr. Kasongo testified that he had been worried when his wife had not returned from the Clinic. (Kasongo 12/13/06.) After receiving a phone call around 5 p.m. that she was at Weiss, he left the children with a neighbor and went to the ER. (*Id.*) He found Ms. Makombe in the waiting room, where she told him of "heat rising from her stomach to her mouth." (*Id.*) She told her husband to go home and care for the children, and that she would be coming home in a couple of days. (*Id.*) He testified that when he left, he did not believe that his wife was going to die. (*Id.*)

Ms. Makombe's condition in fact deteriorated rapidly after her admission to Weiss. (Tornabene 12/11/06; Discharge Summary, 000140.) At around 10:00 p.m. on the night of October 22, a doctor at Weiss called Mr. Kasongo and told him that Ms. Makombe was having difficulty breathing and was in intensive care; when Mr. Kasongo asked to speak to his wife, the doctor informed him that it was impossible. (Kasongo 12/13/06.) On October 24, Weiss doctors told him that Ms. Makombe was on life support, could remain in the same condition for months with no change, and was unlikely to recover. (*Id.*) Tornabene testified that Ms. Makombe was brain dead. (Tornabene 12/11/06.) Mr. Kasongo testified that after speaking with his wife's brother in Belgium, he "took the responsible decision of liberating her." (*Id.*) Falk, Tornabene, and Dr. Katz were present with Mr. Kasongo when Ms. Makombe was taken off the ventilator. (Tornabene 12/11/06; Falk 12/12/06.)

#### **D. Testimony**

As noted, it is undisputed in this case that Ms. Makombe died on October 24, 2001 from

lactic acidosis that was caused by Zerit. (UF ¶¶ 27-28.) The dispute centers around whether the standard of care required the Clinic to have diagnosed her lactic acidosis prior to her coming to the Clinic on October 22, 2001. Plaintiff, to this end, presented the testimony of medical experts and of Ms. Makombe's healthcare providers at the Clinic. The government called two experts. In an effort to support an argument that certain medical records had been altered, Plaintiff also called an expert in the area of document examination. Plaintiff further presented evidence relating to damages, including testimony as to Ms. Makombe's life expectancy and the loss experienced by Mr. Kasongo and his children. The court here presents relevant portions of this testimony.

### **1. Testimony Concerning Document Alteration**

Gerald Richard, a former FBI agent and chief of the FBI document operations and research unit, testified as an expert in the area of document examination. (Richard 12/14/06.) He examined four documents in this case: the October 1 triage form completed by Falk (000047); the October 3 triage form, also completed by Falk (000048); the second page of October 9 treatment notes, written by Tornabene (000050); and Falk's October 22 triage form (000051). He testified that he used techniques including infrared reflectance, infrared luminescence, and dichroic filter examination.<sup>28</sup> (*Id.*)

Richard testified that Falk's writing on the October 1 triage form consisted of two different pen ink formulations. (*Id.*) Specifically, the vital sign entries for blood pressure and pulse rate, and the indication "NAD", were written in a different pen from the entries on the rest of the document. (*Id.*) Similarly, the entries on the October 3 form for blood pressure and SP O<sub>2</sub>, and the "NAD" indication, were written in a different pen from the pen used on the rest of the form; those entries, moreover, were "similar in all observable respects" to the entries written by the second pen on the October 1 triage form. (*Id.*)

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<sup>28</sup> As the government did not contest the techniques used by Mr. Richards, nor his opinions as to the results of his tests, the court will not attempt to describe those techniques here.

The second page of treatment notes from Ms. Makombe's October 9, 2001 appointment, in which Tornabene wrote that she was considering stopping the Zerit but that Ms. Makombe had requested vitamin B6 instead, indeed is dated "10/9." (000050.) Richard testified, however, that the date had been altered, but that the original date was indeterminable because the ink formulations were similar. (Richard 12/14/06.) That ink formulation, which was used on the entire page, was similar to that of the second pen used on the October 1 and October 3 triage forms. (*Id.*)

With respect to the October 22 triage form, Richard testified that three different ink formulations had been used, with the majority of the document written in an ink formulation similar to that of the second pen on the October 1 triage form, the October 3 triage form, and the second page of October 9 treatment notes.

Although the government generally did not dispute Richard's findings, both Falk and Tornabene offered explanations for why certain entries may have been made by different pens. Falk testified that she might, for example, have two pens in her pocket; or she might take a chart to a provider with whom she was consulting, and use a different pen at that time; or she might hand a pen to a patient to fill something out and then use a different pen to continue writing. (Falk 12/12/06.) When asked why the "NAD" indication on the October 1 triage form appears to be written at a different angle from other entries, Falk testified that she remembered taking the form with her when consulting with Boers, who asked her if Ms. Makombe was in distress; when Falk said no, she reached over the desk at which Boers was sitting and wrote "NAD." (*Id.*) Falk denied doctoring the medical records in any way—a denial the court found fully credible.

As for why the differing group of entries may have had similar ink formulations, Tornabene testified that "drug reps" frequently hand out pens at the Clinic; indeed, such pens are "all over the clinic." (Tornabene 12/11/06.) Richard testified that pens from the same manufacturer might have the same or similar ink formulations. (Richard 12/14/06.) From the court's perspective, the most plausible explanation for the alteration of the date on the second page of October 9 treatment notes

is simply that the original entry was mistaken and then corrected. Moreover, any suggestion that that page had been written at a different time, possibly in an attempt to alter the records, is belied by the fact that in the first page of October 9 notes, Falk wrote that Tornabene had indeed instructed her to provide vitamin B6 for Ms. Makombe. (000049.)

Because the evidence does not support any finding that Ms. Makombe's records were doctored or altered after the fact, the court declines to further consider any argument in that regard.

**2. Testimony Concerning the Standard of Care**

**a. Testimony of Clinic Healthcare Providers and Employees**

**i. Mary Tornabene (12/11/06)**

Tornabene began working at the Clinic as an RN in 1990 before obtaining her APN license. She testified that she has received specialized HIV training, has lectured on HIV-related topics including antiretroviral drugs, and completes 16–20 hours per year in continuing education in the HIV field. She has many HIV patients at the Clinic.

Tornabene testified that she knew in 2001 that NRTIs had the known side effect of lactic acidosis. She further testified that the standard of care in 2001 required her to know the signs and symptoms of lactic acidosis, and acknowledged that she, as a treatment provider, was responsible to be on guard for signs and symptoms of potentially fatal side effects of medications.

Tornabene acknowledged that the updated CDC Guidelines, warning of HAART-related lactic acidosis, had been published on August 13, 2001, and that the standard of care required her to know of any such updates. Those guidelines, according to Tornabene, were available on the CDC's website, and on "every HIV treatment web site." All charting stations at the Clinic, Tornabene testified, had binders containing all the latest clinical guidelines, and "everybody knew they were there."

Tornabene further testified as to her knowledge of the specific signs and symptoms of lactic acidosis that she had seen Ms. Makombe beginning to exhibit in August 2001. She acknowledged

that Ms. Makombe's complaints of nausea and GI discomfort on August 10, 2001, were signs and symptoms of lactic acidosis, as were Ms. Makombe's complaints on September 18 of vomiting, abdominal pain in the upper left quadrant, and the fact that her abdomen was large and firm. Similarly, Ms. Makombe's complaints on October 1, 2001 of vomiting, lack of appetite, and mid-epigastric pain were signs and symptoms of lactic acidosis. Tornabene further acknowledged that a complaint of shortness of breath would also be a sign of lactic acidosis; she denied, however, that Ms. Makombe's statement that "respirations are low" signified "shortness of breath." According to Tornabene, "shortness of breath" means "inability to breathe" or to "feel like you are breathing adequately," and a statement that "respirations are low" does not qualify.

Tornabene acknowledged that Ms. Makombe did exhibit shortness of breath on October 3, 2001, which, along with her complaints of weakness, decreased appetite, and vomiting, were signs and symptoms of lactic acidosis. Tornabene testified that Ms. Makombe's additional symptom on October 9 of pain in her legs was also a sign and symptom of lactic acidosis, but that paresthesia, which Ms. Makombe also presented with on October 9, is a "common" side effect of Zerit that has "nothing to do with" lactic acidosis. With respect to Ms. Makombe's reporting on October 9 that her appetite and eating had improved, Tornabene admitted that it was possible that the Ranitidine, which Dr. Katz had prescribed for GERD on October 3, had masked Ms. Makombe's epigastric pain sufficiently that she was able to eat.

As noted, lactic acidosis was on Tornabene's differential diagnosis on October 22, when Ms. Makombe presented with shortness of breath that "feels like suffocating," pain in her ankles and the soles of her feet, lack of appetite, and epigastric pain. She maintained that the standard of care did not require lactic acidosis to have been on the differential diagnosis before then, on October 1 or on October 9. She admitted that when asked in her deposition what else would have been needed on October 1 for lactic acidosis to have been on the differential diagnosis, she had answered, "shortness of breath and leg pain." When asked at trial whether lactic acidosis should

have been on the differential diagnosis on October 9, when Ms. Makombe did exhibit both those symptoms, Tornabene answered, "I wish I had, but no . . . no. She was fine . . . she looked good." Significantly, Tornabene acknowledged that the complaints Ms. Makombe experienced on October 22, which did result in Tornabene's differential diagnosis of lactic acidosis, had been ongoing since at least October 9.

When asked if her process of developing a differential diagnosis consisted of looking at the signs and symptoms and charting possible diagnoses from the most severe to the least severe, Tornabene testified that that was not a "complete definition" of a differential diagnosis; in her deposition, however, she had answered "correct" when asked the question. Tornabene further testified that had lactic acidosis been part of the differential diagnosis before October 22, she would have ordered a serum lactate test, which cost \$20. Tornabene also testified that had she discontinued the Zerit on October 9, the outcome may have been different; however, there was "no guarantee" because other NRTIs (which presumably would have been substituted for Zerit) had the same side effect of lactic acidosis. Ultimately, Tornabene defended her actions by testifying that lactic acidosis is "an incredibly difficult thing to diagnose." She stated that she felt "incredibly awful that this woman got sick and died. She had incredibly good care. She had complaints in her gut. We looked in her gut, we addressed everything that she had . . . ."

**ii. Tammy Falk (12/12/06)**

Falk, an RN, started working at the Clinic in October 2000. Before that, she was a nurse at Illinois Masonic, where she cared for many patients with HIV. Shortly after starting at the Clinic, she attended a training course at the Midwest Aids Training and Educational Center ("MATEC").<sup>29</sup> Falk worked primarily with Tornabene at the Clinic, and as the nurse case manager of the HIV-positive refugee program, had a specific caseload of HIV-positive refugees and their families. Falk

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<sup>29</sup> Boers, who also took the course, explained that it was a one-day, eight-hour course in the care and treatment of HIV and AIDS patients. (Boers 12/11/06.)

testified that her job as a nurse was to identify a patient's signs and symptoms and present those signs and symptoms to a physician or to an APN.

At trial, Falk could not remember whether she knew in October 2001 that lactic acidosis was a side effect of Zerit, but acknowledged that at the time of her deposition, she did not believe she did. She further testified that she did not know in October 2001 that not eating, vomiting, epigastric pain, abdominal pain, muscle pain, and shortness of breath were signs and symptoms of lactic acidosis. She testified that she was not sure whether the standard of care required her to know the side effects of Zerit in 2001; but she admitted that she was required to educate Ms. Makombe about the side effects of Zerit, and that her role in educating patients about their medication including going over common side effects.

Falk acknowledged that the Clinic followed clinical guidelines from the CDC. She testified that those guidelines were available at the nurses' station in the Clinic, but admitted stating in her deposition that she did not know if she ever accessed them there; at trial, she explained that she "[did] them on line" because going to the CDC website was "the most up-to-date way to get those guidelines." She further testified that she did not know in 2001 that the CDC Guidelines indicated that lactic acidosis could be a known side effect of Zerit.

Falk also testified that in 2001, the Journal of the Association of Nurses in AIDS Care, was available on line, and that she had looked up articles pertaining to AIDS and HIV treatment. As noted, that Journal published an article in 2000 that specifically addressed lactic acidosis as a side effect of NRTIs. Falk was not asked at trial whether she had in fact seen the specific article.

**iii. Dr. Marcia Katz (12/13/06)**

Dr. Katz, the medical director of the Clinic in 2001 and a staff physician, testified that she had no independent recollection of Ms. Makombe prior to October 22, 2001. She asserted that Ms. Makombe was not her patient, that Tornabene was Ms. Makombe's primary care provider and was thus "primarily responsible" for her care and treatment, and that she had provided no direct care

with respect to Ms. Makombe until October 22, 2001, when Ms. Makombe was admitted to Weiss.

Dr. Katz was aware in 2001 of case reports of lactic acidosis occurring with patients who were taking Zerit. She testified that vomiting, dizziness, nausea, GI discomfort, upper GI pain, weakness, and shortness of breath are all potential signs and symptoms of lactic acidosis, but that she did not believe that paresthesia is a sign or symptom. Unlike Tornabene, Dr. Katz testified that a statement that “breathing is very low” could be a sign or symptom of lactic acidosis, and that Falk’s indication on the October 1 triage form that Ms. Makombe’s “respirations are low” was a new and different sign and symptom that would make her more suspicious of lactic acidosis.

Dr. Katz acknowledged that Ms. Makombe on October 1, 2001 was exhibiting at least five signs and symptoms of lactic acidosis, including decreased appetite, GI pain, vomiting, low respirations, abdominal pain, and indigestion. In her testimony at trial, she nevertheless answered “no” when asked whether a diagnosis of lactic acidosis should have been made on October 1, whether lactic acidosis should have been on a differential diagnosis, and whether a failure to diagnose lactic acidosis at that time was a deviation from the standard of care. This testimony was inconsistent with her deposition testimony, in which she testified that lactic acidosis could have been diagnosed “at the very earliest” on October 1, answered “correct” when asked if it should have been on the differential diagnosis, and answered “yes” when asked if it was more probably true than not that it was a deviation from the standard of care not to have diagnosed lactic acidosis on October 1. Similarly, Dr. Katz testified at trial that she did not believe that a lactate level test should have been done on October 1, but admitted answering to the contrary in her deposition.<sup>30</sup>

With respect to October 3, when Falk consulted with her, Dr. Katz acknowledged that she did not personally examine Ms. Makombe, and that she never looked at Ms. Makombe’s medical

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<sup>30</sup> When asked in her deposition whether a lactate level test should have been done, Dr. Katz answered “yes—or—” before Plaintiff’s counsel interrupted her. When defense counsel told Dr. Katz to further answer, Dr. Katz declined; and when asked, “so you don’t have anything more to add?” she answered, “correct.”

records. She defended not seeing Ms. Makombe herself, explaining, "I depend on someone to tell me that they think something is different." Moreover, she testified, she would not have ordered a serum lactate test on October 3 because Ms. Makombe's signs and symptoms might also have been signs and symptoms of GERD, PTSD, or HIV-related encephalopathy.

With regard to Falk's role, Dr. Katz testified that nurses at the Clinic make an assessment and decide if a patient is sick enough to require being seen by a physician or APN. Providers at the Clinic trust the nurses' clinical abilities in this regard, according to Dr. Katz, and Katz did trust Falk's abilities. The standard of care did not require Falk to know the signs and symptoms of lactic acidosis in 2001, Dr. Katz opined, because nurses are not expected to know rare complications of medications, and lactic acidosis was "incredibly rare" in 2001. Dr. Katz acknowledged, however, that because Falk was not required to know the signs and symptoms of lactic acidosis, it was "very incumbent" on Dr. Katz to learn, when consulted on October 3, 2001, every sign and symptom that Ms. Makombe presented.

Dr. Katz testified that Ms. Makombe died from lactic acidosis, which was caused by mitochondrial toxicity as a result of Zerit. After Ms. Makombe's admission to Weiss on October 22, according to Dr. Katz, staff at that hospital did everything they could, but the lactic acidosis was too far advanced. Dr. Katz nevertheless opined that she would not have made a diagnosis of lactic acidosis prior to that date.

**iv. Celine Boers (12/11/06)**

Boers, an APN at the Clinic now and in 2001, took classes on the side effects of antiretroviral medications, including Zerit, when obtaining her Ph.D. She also took the MATEC course after getting her Ph.D. in 2000 and before providing care to Ms. Makombe. She acknowledged that the standard of care for an APN for treating a patient on antiretroviral therapy requires knowledge that lactic acidosis is a side effect of Zerit, and of the signs and symptoms.

Boers' only involvement in Ms. Makombe's care occurred on October 1, 2001, when, Falk recalls, Boers gave instructions for Ms. Makombe to take Milk of Magnesia. As noted, Boers has no recollection of consulting with Falk on October 1. Like Dr. Katz, Boers explained that she would not have personally examined Ms. Makombe unless Falk had given her an indication that "something wasn't right."

Boers gave inconsistent testimony as to whether Ms. Makombe's symptoms on October 1, as indicated in Falk's triage form, should have led to a diagnosis of lactic acidosis. She acknowledged that a patient complaining that "respirations are low" is complaining about "a problem breathing," which is known as dyspnea and is a sign of lactic acidosis. On direct examination, Boers testified that based on the October 1 triage form, lactic acidosis should have been on a differential diagnosis on October 1. During the government's cross-examination, however, she testified that even if Falk had told her of all the symptoms on that triage form, she would have done nothing differently, and would not have ordered a serum lactate test because Ms. Makombe's vital signs were normal and did not match a complaint of low respirations. She also testified that she would not have diagnosed lactic acidosis, had she personally examined Ms. Makombe on October 1. But on redirect, she reiterated that even though she does not treat many HIV patients, she could determine, merely by looking at the October 1 triage form, that lactic acidosis should have been part of the differential diagnosis on that date; she further admitted that if lactic acidosis had been on the differential, a serum lactate test should have been done.

Boers further testified that Falk "knows HIV inside and out," and "really appeared to be knowledgeable" about HIV based on the questions she asked patients. Boers testified, however, that she was not aware that Falk did not know the signs and symptoms of lactic acidosis, or that lactic acidosis was a side effect of Zerit.

**v. Heidi Nelson (12/12/06)**

Nelson, the Clinic's Executive Director, testified concerning a conversation with Tornabene and Dr. Katz that occurred when Nelson informed them that this lawsuit had been filed. In that conversation, Tornabene told Nelson that lactic acidosis was not a known side effect of Zerit. Nelson described this conversation in her deposition and testified at that time that she did not recall anything else Tornabene said. At trial, however, Nelson recalled that Tornabene had also said that lactic acidosis was not listed as a side effect of the medications Ms. Makombe was taking; Nelson did not know whether this was a reference to Zerit in particular.

Nelson also testified that as the medical director of the Clinic, Dr. Katz was responsible for making sure that the care providers had the latest CDC guidelines and were aware of any changes, and for determining and conveying to the staff that the August 13 guidelines were to be followed. But, Nelson explained, individual care providers, pursuant to their responsibility to be up to date in their practice area, had an independent obligation to know the information in the August 13 guidelines when those guidelines were published.

**b. Testimony of Plaintiff's Medical Experts**

**i. Sheldon Fields (12/13/06)**

Fields, an RN, APN, and Advanced HIV/AIDS Certified RN, is an assistant professor of nursing at the University of Rochester. He has a Ph.D. in nursing science and teaches and lectures on HIV and AIDS. He also maintains a clinical practice, at a clinic that also happens to be a refugee health center, in which half of his patients have HIV or AIDS. He testified concerning the standard of care for an RN, an APN, and an outpatient clinic in the area of HIV and AIDS.

Fields opined that Falk, Tornabene, Boers, and the Clinic as a whole did not comply with the applicable standard of care. Falk violated the standard of care, Fields testified, because she was unaware of the signs and symptoms of lactic acidosis, and of the fact that lactic acidosis was a side effect of Zerit. Fields noted that lactic acidosis was a well-known, albeit rare, side effect of NRTIs including Zerit, and that an RN working in the AIDS health care field was required to be

familiar with the potentially fatal side effects of antiretroviral medications. Falk, moreover, was required by the standard of care to educate Ms. Makombe on the potential side effects of Zerit, including lactic acidosis.

Furthermore, according to Fields, Falk's role as an RN was to assess a patient's symptoms and, if those symptoms did not "make sense," to report those symptoms to an APN or physician along with relevant medical history. Fields testified that although the standard of care did not require Falk actually to diagnose lactic acidosis, her unfamiliarity with lactic acidosis kept her from accurately conveying symptoms to a provider or assessing what those symptoms may have meant.

Boers violated the standard of care when Falk consulted with her on October 1, 2001, Fields opined, because the information presented in the October 1 triage form should have resulted in placing lactic acidosis on the differential diagnosis. Fields testified that the standard of care required Boers to make further inquiry into Ms. Makombe's medical history, and that if she had in fact seen the triage form, she should have examined Ms. Makombe herself. Fields added that based on his experience working with refugees, a complaint that "respirations are low" should have prompted further inquiry; there is no indication that anyone asked Ms. Makombe what she meant by the comment.

Tornabene violated the standard of care, Fields testified, by failing to appreciate the significance of Ms. Makombe's elevated liver enzymes. Though the elevation was not large, it should have prompted further testing in light of Ms. Makombe's other symptoms. Fields added that Tornabene should have consulted with Dr. Katz if she had any indication that she did not understand those symptoms.

Fields testified that the Clinic as a whole violated the standard of care because Falk, as the only person who saw Ms. Makombe on every visit and as the keeper of her medical history, became the default primary caregiver—a role beyond the scope of her practice as an RN. Moreover, according to Fields, someone at the Clinic should have known that Falk did not know the signs and

symptoms of lactic acidosis. In fact, Fields uses Ms. Makombe's case as an example when teaching undergraduate and graduate students about the responsibilities of nurses and nurse practitioners. He acknowledged that lactic acidosis is extremely difficult to diagnose in its early stages because its symptoms, including vomiting, loss of appetite, weight loss, nausea, and abdominal pain, can have other causes and are not unusual in any patient with AIDS. He noted, however, that Ms. Makombe had other symptoms as well, including shortness of breath beginning on October 1 and leg pain on October 9.

**ii. Dr. Larry Rumans (12/14/06)**

Dr. Rumans, a physician and formerly an Associate Clinical Professor of Medicine at the University of Kansas, practices in Tucson, Arizona and is board-certified in internal medicine and in the subspecialty of infectious diseases. Dr. Rumans began treating HIV and AIDS patients in 1983 in Kansas, and ran the HIV clinic at the Shawnee County Health Agency, which provides treatment for indigent or uninsured patients, until 2006. He has not attended AIDS conferences, does not belong to an HIV or AIDS society, and has neither published nor conducted peer review of articles concerning Zerit and lactic acidosis. When asked how he differed from Dr. Kessler, the government's expert, he characterized himself as a clinician and Dr. Kessler as an academic.

Dr. Rumans testified that he has dealt with lactic acidosis in practice in contexts other than as a side effect of NRTIs, including situations in which patients with infectious diseases have suffered septic shock, in which patients suffer from diabetes, and in which patients developed lactic acidosis as a side effect of other medications. He also treated one patient who had developed lactic acidosis after Dr. Rumans had prescribed Zerit. That patient, who was HIV-positive but did not have AIDS, had lactic acidosis for several weeks but did not die. Dr. Rumans testified that that patient was very different from Ms. Makombe in that the patient's primary complaint was that he was feeling poorly and was "not exactly clear as to what was going on"; the patient saw Dr. Rumans only twice, and on the third visit, Dr. Rumans identified the possibility of lactic acidosis.

As noted, Dr. Rumans considered Dr. Katz, not Tornabene, to be “primarily responsible” for Ms. Makombe’s care and treatment, and thus framed his opinions regarding the standard of care with respect to Dr. Katz alone. He opined that, in her capacity as a care provider, Dr. Katz deviated from the standard of care by failing to timely recognize the signs and symptoms of lactic acidosis, to perform serum lactate testing when such testing became necessary, to timely discontinue the Zerit, to elicit the full extent of Ms. Makombe’s history and presenting symptoms, and to personally examine Ms. Makombe when consulted. He further testified that in her capacity as the medical director of the Clinic, Dr. Katz was responsible for its policies and procedures, and violated the standard of care by failing to train the medical staff to recognize lactic acidosis, to provide appropriate oversight, and to provide continuity in care, with the result that Ms. Makombe’s signs and symptoms were “unappreciated.” Each of these deviations from the standard of care, according to Dr. Rumans, caused or contributed to Ms. Makombe’s death.

Dr. Rumans called Ms. Makombe’s case a “textbook example” of the signs and symptoms of lactic acidosis and its natural progression. Although he acknowledged that there are reports of patients developing lactic acidosis “fairly precipitously and dying very rapidly,” he maintained that the disease typically has a gradual progressive onset, and that patients can be on a drug for three to six months before beginning to develop symptoms. Ms. Makombe, he testified, showed symptoms beginning in August 2001 and progressing over the next two to two-and-a-half months.

Ms. Makombe’s first symptoms, presenting on August 10, 2001, were GI-related. Dr. Rumans testified that the Clinic did not deviate from the standard of care in August 2001, however, because those symptoms were non-specific and difficult to diagnose on initial presentation. But by September 26, 2001, according to Dr. Rumans, Ms. Makombe’s GI symptoms had been persistent since August 10, necessitating more follow-up. Dr. Rumans opined that it was a deviation from the standard of care that lactic acidosis was not part of a differential diagnosis on September 26.

Had lactic acidosis been on the differential, Dr. Rumans testified that the standard of care required a serum lactate test and an electrolyte test. Elevated lactate and low carbon dioxide would indicate lactic acidosis, which could then be confirmed with an arterial blood gas test. A pulse oximeter (SP O<sub>2</sub>) test for oxygenation would be unhelpful, Dr. Rumans explained, because the level of oxygen in the blood “reveals nothing” about acidosis; if a patient complains of shortness of breath, for example, but oxygenation is normal, then a metabolic process is likely responsible for the shortness of breath and further testing is required. Dr. Rumans concluded that because lactic acidosis should have been on the differential diagnosis on September 26, 2001, the failure to run serum lactate or electrolyte tests constituted a violation of the standard of care, as the tests would have indicated lactic acidosis, the Zerit would have been discontinued, and Ms. Makombe would have recovered. The Zerit could have been replaced, according to Dr. Rumans, with other HAART drugs, including protease inhibitors.

Dr. Rumans further testified that the new symptom of shortness of breath on October 1, 2001 provided further support for the conclusion that lactic acidosis should have been part of the differential diagnosis. He concluded that it was a violation of the standard of care for Dr. Katz not to have ordered further testing or to have terminated the Zerit prescription. Had she done so, Dr. Rumans opined, Ms. Makombe would have recovered.

Noting that Falk actually did consult with Dr. Katz on October 3, 2001, Dr. Rumans testified that Dr. Katz violated the standard of care by not personally examining Ms. Makombe on that date, and by not reviewing her chart. He further testified that Dr. Katz should have been able to rule out gallbladder disease on October 3 as the cause of Ms. Makombe’s distress. He explained that the ultrasound of Ms. Makombe’s abdomen, which Dr. Katz had seen, showed that she had gallstones, or cholelithiasis, rather than the more serious condition of gallbladder inflammation, or cholecystitis, which is associated with nausea, vomiting, fever, and chills. Dr. Rumans noted that there was no evidence that Ms. Makombe ever had cholecystitis, and that cholecystitis would not explain her

symptoms in any event: cholecystitis presents with abdominal pain in the upper right quadrant, whereas Ms. Makombe's pain was in the upper left quadrant; and cholecystitis has no relation to shortness of breath, or to Ms. Makombe's later-presenting symptoms of muscle pain and paresthesia. Given that Dr. Katz on October 3 should have ruled out gallbladder disease, Dr. Rumans opined, Dr. Katz should have stopped the Zerit; and had she done so, Ms. Makombe would have recovered.

By October 9, when Ms. Makombe presented with leg pain and paresthesia, Dr. Rumans testified that lactic acidosis provided a "unifying diagnosis," that is, a single diagnosis that could account for all of Ms. Makombe's symptoms. Although he acknowledged that neuropathy in itself is a well-recognized side effect of Zerit, he believed that given the progression of symptoms since August, it was not likely that Zerit was causing a neuropathy independent of lactic acidosis. In any event, he observed, if a neuropathy is severe enough, aside from any consideration of lactic acidosis, then that might be sufficient reason to stop the Zerit in and of itself.

Dr. Rumans also noted that the Ranitidine, which Dr. Katz had prescribed on October 3, alleviates symptoms of GERD by reducing the amount of gastric acid in the stomach. He testified that this could have masked the burning sensation and pain Ms. Makombe was experiencing, thus allowing her to eat, and would explain why she stated on October 9 that her appetite and eating had improved. He concluded that in light of the unifying diagnosis afforded by lactic acidosis, it was a deviation of the standard of care not to have removed the Zerit on October 9, 2001.

Dr. Rumans further testified that the Clinic as a whole deviated from the standard of care. Like Fields, Dr. Rumans believed that Falk's responsibilities went beyond the scope of her practice: she was "the weakest link in the chain," yet she "provid[ed] the greatest degree of care." The Clinic also failed to adequately provide "continuity of care," according to Dr. Rumans, because the care providers did not review Ms. Makombe's medical history. The AST and ALT results were not mentioned on treatment notes from October 3 and October 9, for example, and primary care givers

such as Boers and Dr. Katz were not, when consulted, given information that Ms. Makombe had been seen repeatedly while reporting continuing complaints. Dr. Rumans explained that a patient should be seen by a provider who can appreciate the significance of a series of events historically. He noted, for example, that after Falk consulted with Boers on October 1, a copy of Ms. Makombe's triage form could easily have been placed in Tornabene's mailbox at the Clinic.

Dr. Rumans further noted the significance of the fact that Ms. Makombe came to the Clinic three times without an appointment, with similar complaints, and each time with an additional symptom. In his view, this demonstrated that Ms. Makombe herself knew something was wrong.

**c. Testimony of the Government's Experts**

**i. Dr. Harold Kessler (12/19/06)**

Dr. Kessler is a well-recognized expert in the area of HIV and AIDS treatment. The government elicited testimony from Tornabene and Dr. Katz that they knew of Dr. Kessler and thought him a well-known expert, but that they had not heard of Dr. Rumans prior to this lawsuit; the government's other witness, Bradford Farrington, called Dr. Kessler "world renowned." Dr. Kessler is board-certified in internal medicine and infectious diseases, has received specialized HIV training, and has been involved with the development of the AIDS treatment program at Rush University Medical Center since its inception in the early 1980s. He has participated in national and international symposia in the area of HIV and AIDS, including the International AIDS Congress, and has served on the Board of Directors of the International AIDS Society USA. He testified that he has maintained a clinical practice throughout his medical career, and that his clinical work consists of three half-days at the Rush outpatient clinic and one half-day at the Rush/Cook County HIV clinic, where he supervises HIV care providers.

Dr. Kessler agreed that Zerit caused Ms. Makombe's lactic acidosis, and that lactic acidosis caused her death. He opined, however, that her care and treatment at the Clinic prior to October 22, 2001 did not violate the standard of care. Diagnosis of lactic acidosis is difficult, Dr.

Kessler stated, because many signs and symptoms of the disease, especially early in its course, are GI-related and associated with other common conditions. He thus believed that any delay in diagnosing Ms. Makombe's lactic acidosis was not a deviation from the standard of care.

Dr. Kessler based his opinion on a review of medical literature in 2000 and 2001, containing reports of lactic acidosis in which the length of time between the manifestation of symptoms and a diagnosis of lactic acidosis ranged from one week to three months, and on his own experience with a single patient who had lactic acidosis. His review of the literature relied on an abstract of a 1999 article from the FDA, discussing case reports of lactic acidosis associated with NRTIs through June 1998,<sup>31</sup> and a Spanish study that discussed twelve cases of NRTI-related lactic acidosis. (Def.'s Ex. 11.) Dr. Kessler testified that the incidents of lactic acidosis in the FDA abstract showed a range of one to six weeks from the onset of symptoms to a diagnosis of lactic acidosis, and that the cases discussed in the Spanish study showed a range of 24 hours to as much as three months. Upon cross-examination, Dr. Kessler admitted that neither article revealed the progression of lactic acidosis in each patient, how many times the patient was examined, which signs and symptoms came first, or which symptoms presented at what time during the period before diagnosis. Moreover, Dr. Kessler acknowledged that only one patient in the Spanish study was diagnosed as long as three months after presenting with symptoms, which included vomiting, weight loss, and numbness; the remaining eleven were diagnosed from 24 hours to one month after manifesting symptoms. One of the patients who was diagnosed in 24 hours presented with abdominal pain only; another was diagnosed just five days after presenting with nausea and abdominal pain; and another, diagnosed in one week, had only nausea and vomiting. Dr. Kessler further admitted that in his review of the literature, he had found no patient profile matching that of Ms. Makombe.

With respect to his own patient with lactic acidosis, Dr. Kessler testified that the patient

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<sup>31</sup> Dr. Kessler noted that the FDA article is what prompted the insertion of warnings of lactic acidosis in the labeling of NRTIs, including Zerit, in 1999.

began to manifest symptoms in August 2000 but was not diagnosed with lactic acidosis until October 2000. Dr. Kessler saw the patient three times during those three months. In the first and second visits, the patient's only major symptom was decreased appetite. The patient presented with abdominal discomfort, increased abdominal girth, and shortness of breath in the third visit in October 2000; Dr. Kessler testified that it was not until the patient reported shortness of breath that "the light bulbs went off" and he hospitalized the patient with suspicion of lactic acidosis.

Dr. Kessler explained that lactic acidosis is not often diagnosed until there are respiratory symptoms such as rapid breathing or dyspnea, which he described as the sensation of shortness of breath. He nevertheless opined that lactic acidosis was not required to be part of the differential diagnosis<sup>32</sup> on October 1, 2001, when Ms. Makombe stated "respirations are low." Dr. Kessler testified that he did not know what "respirations are low" means, although he admitted that the complaint needed follow-up. He also testified that an SP O2 oxygenation test was unnecessary on October 1 because Ms. Makombe's respiration rate (22) did not show "rapid breathing."

Nor did Dr. Kessler believe that the standard of care required lactic acidosis to be on the differential diagnosis on October 3, when Falk in fact noted on the triage form that Ms. Makombe complained of "shortness of breath." He testified that the SP O2 test, showing a 98% oxygenation rate, demonstrated that Ms. Makombe had enough oxygen in her blood. Significantly, however, he conceded that oxygenation rate has nothing to do with lactic acidosis, and that patients with lactic acidosis caused by Zerit have normal oxygen levels in both blood and tissue.

Dr. Kessler further admitted on cross-examination that it was clear by October 3, 2001 that based on the ultrasound results, Ms. Makombe did not have cholecystitis and her symptoms were not caused by gallstones. Like Dr. Rumans, he noted that Ms. Makombe's September 18 complaint

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<sup>32</sup> Dr. Kessler testified that a differential diagnosis lists the most severe, or life-threatening possibility at the top, and that that potential diagnosis should be ruled out, and so on down the list. He added, however, that the list should start with the most common complications, and that "it takes a long time to get down to rare complications."

of upper abdominal pain in the upper left quadrant was inconsistent with gallbladder disease, which would present with pain in the right or middle of the abdomen. Dr. Kessler nevertheless concluded that when Falk consulted with Dr. Katz on October 3, Dr. Katz had no obligation to examine Ms. Makombe, to follow up on the ultrasound, or even to look at Ms. Makombe's medical chart, because Dr. Katz was not Ms. Makombe's primary care provider. Even if Dr. Katz had seen Ms. Makombe or examined her chart on October 3, according to Dr. Kessler, it would not change his opinion with regard to the difficulty in diagnosing lactic acidosis.

Finally, Dr. Kessler agreed with Dr. Rumans that lactic acidosis would have provided a unifying diagnosis on October 9, 2001, when Ms. Makombe presented with leg pain and paresthesia, but again opined that a failure to have lactic acidosis on a differential diagnosis on that date did not violate the standard of care. He noted that paresthesia or neuropathy was a well-known side effect of Zerit independent of lactic acidosis. He acknowledged that the 1999 package insert for Zerit states that Zerit should be removed if a patient develops signs of a peripheral neuropathy;<sup>33</sup> but Dr. Kessler disagreed with that instruction, explaining that in his opinion, the drug could be continued, even at the same dose, if the neuropathy did not progress. For that reason, according to Dr. Kessler, the fact that Ms. Makombe may have developed a peripheral neuropathy on October 9 did not provide an independent reason for stopping the Zerit.

**ii. Bradford Farrington (12/15/06)**

Farrington is an RN, an APN, and an Advanced HIV/AIDS Certified RN. He currently is a research assistant, nurse practitioner, and clinical team leader at Wayne State University in Detroit. He testified that the vast majority of his work is in clinical practice, and that since 1987, he has

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<sup>33</sup> The package inserts for Zerit in 1999, 2000, and 2001, as reproduced in the PDR, state the following: "Patients should be monitored for the development of peripheral neuropathy, which is usually characterized by numbness, tingling, or pain in the feet or hands. . . . If these symptoms develop on treatment, stavudine therapy should be interrupted." (1999, 2000, 2001 PDR, Pl.'s Ex. 50-52.)

focused on the care of HIV and AIDS patients. He shares a practice with a physician, with whom he has a collaborative agreement, and considers himself to be his patients' primary care provider.

Farrington testified from personal experience with lactic acidosis: in late 2001, he himself developed lactic acidosis as a side effect of the diabetes drug Metformin. Between September and December 2001, he experienced episodic shortness of breath, vomiting, and abdominal pain. He ultimately suggested a serum lactate test to his doctor, and based on the results, went to the hospital. Farrington also had one patient on Zerit who developed lactic acidosis, and who had presented with abdominal pain, shortness of breath, and weight gain.<sup>34</sup> The patient was ultimately hospitalized because she "looked awful" and had developed "rapid shallow breathing." Lactic acidosis was not on the differential diagnosis at the time, Farrington testified, because this occurred in 1996 and there were as of that time no reports linking lactic acidosis and Zerit. On cross-examination, he admitted to testifying in his deposition that he last saw the patient a month before the diagnosis of lactic acidosis, and that he could not recall any of the patient's symptoms during that month.

Farrington testified that lactic acidosis is not a common problem and thus is "low on the radar." He described the disease's signs and symptoms as "very vague," and suggestive, in the early stages, of conditions such as indigestion, GERD, or viral flu. Alone among the testifying experts, he stated that the shortness of breath associated with lactic acidosis "usually [is] the result of abdominal pain." Describing the risk factors for lactic acidosis, Farrington testified that practitioners use the term "fair, fat, and 40s" to describe woman at risk for both lactic acidosis and gallbladder disease. He also testified, however, that the "vast majority" of the initial case reports of NRTI-related lactic acidosis involved female, obese African-Americans over the age of forty.

Farrington opined that the standard of care was not breached by Falk, Boers, Tornabene,

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<sup>34</sup> Neither the CDC guidelines nor the testimony of any other witness indicates that weight *gain* is a sign of lactic acidosis; to the contrary, weight *loss* is a recognized symptom.

or the Clinic. He testified that Clinic providers, in investigating gallbladder disease, were following a diagnostic plan that was supported by clinical evidence. A diagnosis of lactic acidosis became appropriate for the first time on October 22, according to Farrington, because on that date, Ms. Makombe “looked awful.”

Farrington’s opinions did not fare well upon cross-examination. He admitted to testifying in his deposition that the reason the standard of care did not require lactic acidosis to be part of the differential diagnosis before October 22, 2001 was that there were no “black box” warnings on the package inserts for Zerit until 2002. He acknowledged at trial that he had been mistaken, and that the package inserts in 1999, 2000, and 2001 indeed included such warnings. He further admitted that as far back as 1998, there were lectures and published anecdotal reports of lactic acidosis associated with Zerit. Finally, he admitted in his testimony that the standard of care in 2001 required an RN to know the signs and symptoms of lactic acidosis, and to know that lactic acidosis was a side effect of Zerit.

### **3. Testimony Relating to Life Expectancy and Damages**

#### **a. Dr. Richard Novak (12/14/06)**

Dr. Novak, a Professor at University of Illinois at Chicago, is board-certified in internal medicine and infectious diseases. He is the director of the UIC HIV/AIDS project, a network of eight community-based clinics throughout Chicago that provide HIV primary care to mostly indigent minority patients. He testified only as to Ms. Makombe’s life expectancy.

Dr. Novak testified that had Ms. Makombe not died from lactic acidosis, it is more probably true than not that she would have enjoyed a normal life expectancy. He based that opinion on a review of the literature and on his experience treating HIV and AIDS patients. He testified that most published studies were not relevant because they addressed the natural progression of AIDS and HIV, assuming no treatment. He did find one relevant study, a meta-analysis that utilized data from thirteen other studies, involving 9,000 patients in Europe and North America. The study predicted

patients' probabilities of survival based on their response to HAART, and concluded that the best predictor of survival is a patient's response to treatment six months after initiating antiretroviral therapy.

Dr. Novak acknowledged the study's limitations: it predicted survival rates for only three-and-a-half years following the initiation of antiretroviral therapy, and did not allow for future developments in HIV treatment. Because of these limitations, Dr. Novak testified that he also based his opinions on his twenty years of experience treating patients with HIV. He explained that with HAART, HIV is no longer a death sentence; rather, it is now a "chronic disease" similar to diabetes, that "can be managed for an indefinite period of time."

Based on his experience and the study, he considered four factors in determining Ms. Makombe's life expectancy, had she not died of lactic acidosis: lab values, including CD4 (T-cell) count and viral load; the presence of AIDS-defining illness; compliance with medications; and clinical response. Dr. Novak explained that according to these criteria, Ms. Makombe's prognosis was good. Her CD4 count had increased from 48 to 112 in six months, and her viral load became undetectable. She had never experienced an AIDS-related illness. Dr. Novak testified that Ms. Makombe must have been adherent to the antiretroviral drugs, because had she stopped taking her medication, her viral load would have risen within one or two weeks. Finally, Ms. Makombe appeared to respond well clinically, gaining weight and reporting more energy.

According to Dr. Novak, patients in the study with Ms. Makombe's lab results had a 97.5% probability of surviving over the three-and-a-half year span of the study. Beyond that time, in light of Ms. Makombe's response to HAART, Dr. Novak opined that Ms. Makombe would have continued to do well for an indefinite period of time, and that there was no reason for her not to have a normal life expectancy.

On cross-examination, the government confronted Dr. Novak with the expert report he had prepared for this litigation, in which he stated that a "conservative estimate" of Ms. Makombe's life

expectancy would be “at least a 50% likelihood of surviving 10 years, had she continued on potent antiretroviral therapy,” and assuming no new advancements in treatment of HIV within that ten-year period. (Novak Report, Pl.’s Ex. 44.) Dr. Novak also admitted that when asked in his deposition “how firm” was his projection that Ms. Makombe would have lived ten years, he answered, “It’s more likely than not that that would have happened.” He explained at trial, however, that his previous statements did not mean that 50% of patients like Ms. Makombe would in fact die after ten years. He pointed out that he had also testified in his deposition that it was quite likely that Ms. Makombe would not die of an HIV-related condition.

Following Dr. Novak’s testimony, Plaintiff asked the court to take judicial notice of life expectancy tables published by the federal government. Those tables indicate that the life expectancy of a black female born on April 24, 1962 is eighty-one years.

**b. Dr. Rumans’ Testimony Regarding Life Expectancy**

A portion of Dr. Rumans’ testimony was directed to Ms. Makombe’s life expectancy. Like Dr. Novak, Dr. Rumans testified that adherence to medication and a lack of opportunistic AIDS infections are positive factors with regard to a patient’s future survival. Ms. Makombe’s CD4 count, reduction in viral load, compliance with medications, and lack of a history of opportunistic infections all “bode well for the future.” In addition, Dr. Rumans explained, newer classes of HIV drugs have appeared since 2002, as well as improvements in existing HAART medications.

Dr. Rumans also testified that had the Zerit been discontinued, Ms. Makombe could have taken other HIV drugs, including protease inhibitors, as part of her HAART regimen. He opined that “conservatively speaking,” a survival prediction of ten years was “reasonable” had Ms. Makombe switched to protease inhibitors. He added that he had seen fifteen to twenty years’ survival rates with HIV, given drug compliance, and it was “not unreasonable” for an individual to expect a virtually normal life expectancy despite having HIV. When confronted with a statement from his 2002 deposition that Ms. Makombe had a ten years’ life expectancy without any advancement in

medicine, Dr. Rumans explained that there had been advancements just since he made that statement, and that with those advancements, Ms. Makombe could live for fifteen or twenty years; and with further advancements, "maybe longer."

**c. Innocent Kasongo (12/13/06)**

Mr. Kasongo testified that his wife began experiencing nausea and indigestion in August 2001, and that her symptoms continued to grow through October. He described Ms. Makombe's health as deteriorating day by day during that time. He testified that he "had to watch her suffer."

He described his loss as "very great" and the damage from his wife's death "inestimable." He testified that he had "lost a treasure," and that he felt like the "sky had fallen down" on his head. He described his wife as a "marvelous woman," the "foundation of our household," and his "advisor." He testified that Ms. Makombe was "the support of [his] children" and their "guide" who provided "all the affection that [his] children had."

Mr. Kasongo testified that his children were traumatized by his wife's death, and that the family is in counseling. Sara, the youngest child, has woken several times during the night and asked her father to open the door because "mama is outside the door." She has also interrupted classes at school, demanding that Mr. Kasongo come to the school to talk to her. Moises, the oldest child, had been very active before his mother's death; after his mother's death, according to Mr. Kasongo, Moises became "closed in upon himself" and withdrawn, and began drinking and smoking with teenagers from the neighborhood. After Mr. Kasongo found him with marijuana, Moises ran away from home for two days.

Immediately after Ms. Makombe's death, Mr. Kasongo stayed home with the children rather than go to work. He explained that "circumstances dictated" this decision because his children needed him at home. Eventually, Mr. Kasongo returned to work as a receptionist, hiring a woman to stay with the children while he was at work. Hoping to remove Moises from a negative environment, Mr. Kasongo then moved the family to Skokie, where he obtained a security job close

to home and where Moises entered a drug treatment program.

### **DISCUSSION**

The Federal Tort Claims Act provides a remedy for personal injury or death caused by the negligent or wrongful act or omission of government employees while acting within the scope of employment. See 28 U.S.C. § 1346(b)(1). The federal government is liable in the same manner and to the same extent as a private individual in accordance with the law of the state where the cause arose, which in this case is Illinois. See 28 U.S.C. §§ 1346(b)(1), 2671, & 2674; *Midwest Knitting Mills, Inc. v. United States*, 950 F.2d 1295, 1297 (7th Cir. 1991). It is undisputed that at all relevant times, and for purposes of coverage under the Act for the allegations in this lawsuit, the Clinic and its employees have been deemed employees of the United States. (UF ¶¶ 5, 9-10.)

Plaintiff brings negligence claims pursuant to Illinois' wrongful death statute, 740 ILCS 180/1 *et seq.*, (Compl. Count I ¶¶ 26-27), and under the Illinois survival statute, 755 ILCS 5/27-6. (Compl. Count II ¶¶ 24-26.) The wrongful death claim, which Plaintiff brings on behalf of himself and of his and Ms. Makombe's three children, alleges loss of society and loss of consortium as a proximate result of the negligence of the Clinic and its employees. (Compl. Count I ¶¶ 25-26.) The survival action alleges that Ms. Makombe suffered extreme conscious pain and suffering before her death. (Compl. Count II ¶¶ 24.) Plaintiff seeks an award of \$4 million for the wrongful death claim, and \$2 million for the survival action. (Damages Brief ("Pl.'s Br."), at 10, 13.)

#### **I. Negligence/Wrongful Death**

The Illinois Wrongful Death Act, 740 ILCS 180/1, provides an independent cause of action for damages arising from a decedent's death caused by wrongful act, neglect or default. *Kessinger v. Grefco, Inc.*, 251 Ill. App. 3d 980, 982, 623 N.E.2d 946, 948 (4th Dist. 1993) (citations omitted). The Act's purpose is to compensate a surviving spouse and next of kin for the pecuniary losses sustained as a result of the decedent's death. *Id.* (citing *In re Estate of Finley*, 151 Ill. 2d 95, 101, 601 N.E.2d 699, 701 (1992)). "Aside from the additional element of the occurrence of death,

the elements of a wrongful death claim are identical to those of a common law negligence claim.” *Williams v. Manchester*, 372 Ill. App. 3d 211, 223, 864 N.E.2d 963, 974 (1st Dist. 2007). In a medical malpractice case based upon negligence, the burden is on the plaintiff to prove (1) the proper standard of care by which a physician’s conduct may be measured; (2) a negligent failure to comply with the applicable standard; and (3) a resulting injury proximately caused by the physician’s lack of skill or care. *Donais v. United States*, 232 F.3d 595, 598 (7th Cir. 2000) (citing *Purtill v. Hess*, 111 Ill. 2d 229, 241-42, 489 N.E.2d 867, 872 (1986) (citations omitted)).

This case turns on whether the Clinic and/or its medical staff breached the applicable standard of care. Plaintiff contends that in light of the standard of care as established by the expert testimony of Sheldon Fields and Dr. Rumans, Ms. Makombe’s symptoms warranted a diagnosis of lactic acidosis at least as early as September 26, 2001. According to Plaintiff, the failure of the Clinic’s medical staff to diagnose lactic acidosis upon Ms. Makombe’s visiting the Clinic on that day, or upon her visits on October 1, October 3, and October 9, 2001, and the staff’s consequent failure to discontinue the Zerit, was a breach of the standard of care that proximately caused Ms. Makombe’s death on October 24, 2001. (Pl.’s Closing Argument 12/19/06.) The government maintains that lactic acidosis is an exceedingly rare complication of Zerit, and that the condition is very difficult to diagnose because its signs and symptoms are intermittent, non-specific and suggestive of other conditions. Relying on Dr. Kessler’s testimony, the government asserts that a diagnosis of lactic acidosis simply “takes a long time” and is timely if made up to three months after a patient begins exhibiting symptoms. (Def.’s Closing Argument 12/19/06; Def.’s Br., at 1.) The government takes the position that the diagnosis of Ms. Makombe’s lactic acidosis on October 22, 2001, coming less than three months after she began presenting symptoms on August 10, 2001, thus did not deviate from the standard of care. The government advances no arguments concerning proximate cause.

**A. Breach of the Standard of Care**

In a medical malpractice case, the standard of care against which a defendant's conduct is measured is not the highest degree of skill possible, but the reasonable skill that a physician in good standing in the community would use in a similar case. *Newell v. Corres*, 125 Ill. App. 3d 1087, 1094, 466 N.E.2d 1085, 1090 (1st Dist. 1984). Unless the physician's negligence is so grossly apparent or the treatment so common as to be within the everyday knowledge of a layperson, expert medical testimony is required to establish the standard of care and the defendant physician's deviation from that standard. *Purtill*, 111 Ill. 2d at 241-42, 489 N.E.2d at 872. The weight given to expert medical expert testimony is for the trier of fact to determine. *Suttle v. Lake Forest Hosp.*, 315 Ill. App. 3d 96, 103, 733 N.E.2d 726, 732 (1st Dist. 2000).

The court concludes that the Clinic and its employees breached the standard of care in failing to timely diagnose Ms. Makombe with lactic acidosis. Lactic acidosis, although rare, was in 2001 a known side effect of Zerit. Ms. Makombe began demonstrating signs and symptoms of lactic acidosis on August 10, 2001, and continued to develop additional signs and symptoms over the course of the next two months. As more fully discussed below, the court finds, based on a review of the expert testimony and the evidence in this case, that the standard of care required Clinic staff to suspect, and test for, lactic acidosis by October 1, October 3 or October 9, 2001 at the latest.

#### **1. The Government's View of the Standard of Care is Untenable**

As a preliminary matter, the court finds unpersuasive the government's position that Zerit-related lactic acidosis is so rare, and a diagnosis so difficult, that the standard of care with respect to diagnosing lactic acidosis is not violated so long as a patient is diagnosed within three months of the initial presentation of symptoms. (Def.'s Br., at 1.) While it is undisputed in this case that lactic acidosis is rare, Plaintiff, as noted, presented uncontradicted evidence that it was nonetheless a well-known side effect of Zerit by 2001 and probably for several years before that. Dr. Rumans testified that lactic acidosis had been linked to AZT as far back as 1989, and was a known side

effect of NRTIs by the time Zerit arrived in 1994. Reports of a link between lactic acidosis and Zerit appeared as early as 1996, and by 2000 there were warnings in the medical literature and at HIV care provider meetings. By 2001, according to Dr. Rumans, the link between Zerit and lactic acidosis had been clearly established within the community of AIDS and HIV treatment providers.

The government's expert, Dr. Kessler, conceded that there had been a "spike" in Zerit-related lactic acidosis in 1997. He further testified that the abstract published by the FDA in 1998, upon which he partly based his opinions, reported cases of lactic acidosis as a side effect of NRTIs and prompted the use of warning labels in the packaging of Zerit. Indeed, a "black box" warning, noting reported cases of fatal lactic acidosis with the use of Zerit, appeared at the top of the FDA-approved package inserts for Zerit in 1999, 2000, and 2001. In addition, the CDC Guidelines specifically warned of HAART-related lactic acidosis throughout 2001, with an update appearing on August 13, 2001—right around the time Ms. Makombe began showing symptoms. In the court's view, any argument that the standard of care should be relaxed, due to the rarity of lactic acidosis, is overcome by the fact that the condition was a well-recognized side effect of Zerit in 2001.

Moreover, because lactic acidosis is so often fatal, it is precisely the type of side effect that a treating physician is obliged to consider. In Ms. Makombe's case, that was doubly so because she fit the profile of a patient at risk for lactic acidosis. The package inserts for Zerit in 1999 and 2000 noted that the majority of reports of lactic acidosis involved women, and the 2001 version listed obesity and prolonged NRTI exposure were additional risk factors. Farrington, who testified on behalf of the government with respect to the standard of care for RNs and APNs, testified that the "vast majority" of initial case reports of NRTI-related lactic acidosis involved female, obese African-Americans over the age of forty. Ms. Makombe, an African woman who weighed 194 lbs. in August 2001 and was thirty-nine years old, was thus at risk.

Finally, the court rejects any notion that the standard of care for diagnosing lactic acidosis should be defined exclusively on a temporal basis. As noted, the government contends that

because lactic acidosis is so difficult to diagnose, due to the non-specific nature of the symptoms, a delay in diagnosing the condition does not violate the standard of care as long as a diagnosis is made within three months of a patient's initial symptoms. In support of this position, the government relies on the testimony of Dr. Kessler, who arrived at the three-month time frame by noting reports in the medical literature—the FDA abstract noted above, and a Spanish study of twelve cases—of patients who had been diagnosed anywhere from twenty-four hours to as long as three months after first presenting with symptoms of lactic acidosis. But these reports barely support Dr. Kessler's conclusion, as he admitted upon cross-examination that only one of the patients in the Spanish study was diagnosed after three months; the remainder were diagnosed within a one-day to one-month range, and the patients in the FDA abstract were all diagnosed in a one- to six-week period. Moreover, some of the patients who had been diagnosed in a very short time showed far fewer symptoms than Ms. Makombe, including one patient who was diagnosed in twenty-four hours despite presenting with abdominal pain only, and another who had only nausea and vomiting yet was diagnosed in a week. Neither study revealed the progression of the patients' symptoms, including which symptoms appeared when and in what order, or how many times the patient was examined.

More fundamentally, the court is troubled by any definition of the standard of care that uses a nearly arbitrary time limit, and only that time limit, to determine whether a breach of duty has occurred. All the physicians and nurse practitioners in this case testified that a diagnosis of lactic acidosis is made according to the signs and symptoms presented by the patient. Yet the government's view of the standard of care is effectively divorced from the consideration of those signs and symptoms. Indeed, the government's discussion in closing arguments of the difficulty of diagnosing lactic acidosis prompted the court to inquire whether the government was taking the position that a failure to diagnose lactic acidosis in 2001 could not, as a matter of law, deviate from the standard of care. The government denied making this argument, but confirmed its view that

diagnosing lactic acidosis simply “takes a long time,” and that the standard of care would be violated only if a diagnosis were made “four months or five months” from the initial presentation of symptoms. The court finds this approach to determining the parameters of the standard of care unsatisfactory, and therefore declines to adopt the government’s definition of the standard of care for diagnosing lactic acidosis.

## **2. Lactic Acidosis Should Have Been Suspected On October 1, 2001**

As noted, the nature of the signs and symptoms of HAART-related lactic acidosis is undisputed in this case. According to the CDC Guidelines, the initial signs and symptoms include GI-related symptoms, dyspnea, abdominal pain or distention, nausea, vomiting, weight loss, anorexia or lack of appetite, and weakness. Dr. Rumans testified that lactic acidosis begins presenting with GI symptoms that may include nausea, vomiting, loss of appetite, weight loss, and abdominal discomfort ranging from pain to an unpleasant feeling of fullness and distension. Inadequate respiration, including that a patient may complain of feeling breathless, occurs next, followed by complaints of fatigue or weakness, muscle aches and pains, and numbness and tingling in the extremities. The government did not challenge Dr. Rumans’ description of the progression of the disease, nor present any evidence inconsistent with it.

Dr. Rumans testified that Ms. Makombe’s case was a “textbook example” of the presentation of signs and symptoms of lactic acidosis and of the disease’s natural progression, and Ms. Makombe’s medical records support this characterization. She presented with nausea and GI discomfort on August 10, 2001, and complained of continued GI distress on August 28. On September 18, she reported vomiting and abdominal pain in the upper left quadrant, and her abdomen was large and firm. On September 26, she again complained of vomiting. October 1 brought complaints of lack of appetite, abdominal pain, vomiting, and a remark that “respirations are low.” On October 3, Falk noted shortness of breath, weakness, and dizziness in addition to the ongoing complaints of vomiting and decreased appetite. On October 9, the list of Ms. Makombe’s

symptoms grew to include leg pain and numbness, and a twelve-pound weight loss since August. When Tornabene finally put lactic acidosis on the differential diagnosis on October 22, 2001, Ms. Makombe presented with shortness of breath that “feels like suffocating,” “burning” epigastric pain, severe pain in her ankles and feet, vomiting, dizziness, weakness, and increased abdomen size.

In light of these signs and symptoms, and their progression, the court concludes that the standard of care required lactic acidosis to have been suspected well before October 22. Based on the court’s review of the evidence, October 1, 2001 is the date upon which lactic acidosis should, at a minimum, have been part of a differential diagnosis for purposes of ruling it out.

Prior to that date, Ms. Makombe’s symptoms were mostly GI-related and non-specific. Her symptoms on August 10 and August 28, 2001 consisted merely of nausea and GI discomfort and distress;<sup>35</sup> and she reported on August 28 that the GI distress was aggravated by spicy foods and alleviated by antacids, further supporting a diagnosis of a GI-related condition. Moreover, her weight of 194 lbs. on August 28 represented a significant gain from previous readings of 179 lbs. in March 2001 and 153 lbs. in September 2000. Tornabene testified that the weight gain was a “good thing,” and weight gain indeed runs counter to the signs and symptoms of lactic acidosis.

Ms. Makombe’s symptoms on September 18 and 26, 2001 were also GI-related and non-specific: vomiting and abdominal pain. The court acknowledges that Dr. Rumans testified that despite the non-specific nature of Ms. Makombe’s symptoms up until this point, lactic acidosis should have been part of the differential diagnosis on September 26 because the persistence of these GI-related symptoms should have prompted further investigation. Although the court generally found Dr. Rumans’ testimony credible and persuasive, the court is not satisfied that Plaintiff has proved by a preponderance of the evidence that the standard of care required a

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<sup>35</sup> Beginning on August 10, 2001, Ms. Makombe also began complaining of fullness and tenderness in her breasts; those complaints continued in intermittent fashion through October 22. Neither party has attached significance to this symptom, either as an indication or a contraindication of lactic acidosis.

diagnosis of lactic acidosis based only on the symptoms Ms. Makombe had presented prior to and including September 26. No other witness testified that lactic acidosis should have been diagnosed on that day, and those symptoms indeed are suggestive of many commonplace conditions.

In contrast, a good deal of evidence, including the testimony of Ms. Makombe's own care providers and the government's own expert, Dr. Kessler, supports Dr. Rumans' conclusion that lactic acidosis should have been suspected on October 1, 2001 in any event. On that day, in addition to GI-related complaints of "no eating," vomiting, and abdominal pain, Ms. Makombe told Falk "respirations are low." As noted, shortness of breath and dyspnea are recognized signs and symptoms of lactic acidosis. Significantly, both Dr. Rumans and Dr. Kessler testified that dyspnea refers not only to actual shortness of breath, but to the sensation of shortness of breath.

Tornabene testified, however, that the statement "respirations are low" does not amount to a complaint that Ms. Makombe did not feel that she was breathing adequately. The court cannot agree. As a general matter, a comment that "respirations are low" is indeed suggestive of a complaint concerning one's breathing; indeed, it is difficult to imagine any other interpretation of such a complaint from a layperson. In this case, particularly as the remark came from a patient who spoke English as a second language and was not accompanied by an interpreter, the comment should at the very least have prompted further inquiry into what Ms. Makombe meant. Professor Fields, testifying for Plaintiff as to the standard of care for nurses and APNs, opined that based on his own experience working with refugees, a statement that "respirations are low" requires investigation. Yet neither Falk, who examined Ms. Makombe on October 1, nor Boers, with whom Tornabene consulted, asked Ms. Makombe what she meant by the comment; and Falk did not mark the box labeled "shortness of breath" on the triage form. The court concludes that Ms. Makombe in fact complained of dyspnea, a recognized sign and symptom of lactic acidosis, on October 1, 2001.

Significantly, Plaintiff's care providers themselves admitted that lactic acidosis should have

been part of a differential diagnosis as early as October 1. Boers explicitly testified that based just on the October 1 triage form—which she does not remember seeing, although Falk testified that she showed it to Boers—lactic acidosis should have been on the differential. Dr. Katz acknowledged that Ms. Makombe was exhibiting at least five signs and symptoms of lactic acidosis on that date, including decreased appetite, vomiting, low respirations, abdominal pain, and indigestion. She testified that a statement “breathing is very low” could be a symptom of lactic acidosis, and that Ms. Makombe’s comment that “respirations are low” would indeed make her suspicious of lactic acidosis. Although she opined that the standard of care did not require a diagnosis of lactic acidosis on October 1, she admitted testifying in her deposition that lactic acidosis could have been diagnosed “at the very earliest” on October 1, that lactic acidosis should have been on the differential diagnosis on that date, and that it was a deviation from the standard of care not to have diagnosed lactic acidosis at that time.

Furthermore, Dr. Kessler, while opining that the standard of care was not breached in this case, nonetheless emphasized the importance of shortness of breath to a diagnosis of lactic acidosis. Discussing his patient who had developed lactic acidosis, and who had presented with abdominal discomfort, increased abdominal girth, and decreased appetite in two visits over two months, Dr. Kessler testified that “the light bulbs went off” when the patient presented with shortness of breath on the third visit. Indeed, he further testified that a diagnosis of lactic acidosis is often not made until the patient shows respiratory symptoms such as rapid breathing or dyspnea. Like Tornabene, he did not equate “respirations are low” with these respiratory symptoms; as noted above, the court finds this implausible.

Given the constellation of symptoms that Ms. Makombe had been exhibiting since August 10, 2001—nausea, vomiting, abdominal and GI pain, lack of appetite, and now shortness of breath—the “light bulbs” should have gone off on October 1. Plaintiff points out, however, that no care provider was actually aware of all these symptoms, or their duration, on October 1. As

noted, Falk consulted with Boers on that day. According to Falk, she showed Boers the October 1 triage form, which indicated complaints of “no eating,” abdominal pain, vomiting, constipation, and the comment about low respirations. Boers, who gave instructions that Ms. Makombe take Milk of Magnesia for the constipation, testified that she did not remember the consultation or ever seeing the form. The fact that Boers further testified that she would be unlikely to prescribe Milk of Magnesia for a patient who was vomiting, suggests either that Falk did not in fact show her the form or otherwise describe Ms. Makombe’s symptoms, or that Boers disregarded symptoms other than constipation. Even if Boers did see the form and did consider all the symptoms indicated therein, Boers did not review Ms. Makombe’s medical chart, and thus had no idea that many of her GI symptoms had been ongoing since August 10.

Fields testified that not only was lactic acidosis required to have been on the differential diagnosis on October 1, but Boers’ failure to review Ms. Makombe’s medical history or to examine her personally constituted violations of the standard of care. However compelling this opinion may be, the court need not determine whether Boers deviated from the standard of care by not personally examining Ms. Makombe or reviewing her chart. As Boers admitted in her testimony, lactic acidosis should have been on the differential diagnosis, based the October 1 triage form alone; no further physical examination was needed, and while a review of Ms. Makombe’s chart would have provided a further reason to suspect lactic acidosis, the signs and symptoms she was exhibiting on October 1 were sufficient in themselves.

Once lactic acidosis is part of a differential diagnosis, Dr. Rumans testified, the standard of care requires a serum lactate test and an electrolyte test. If lactate is elevated and carbon dioxide levels low, lactic acidosis is confirmed with an arterial blood gas test. This point appears undisputed; indeed, Tornabene also testified that she would have ordered a serum lactate test, had lactic acidosis been on a differential diagnosis prior to October 22. Both Dr. Kessler and Dr. Rumans testified that routine monitoring of serum lactate levels is not required, as patients taking

NRTIs can have elevated lactate without developing lactic acidosis. Once lactic acidosis is suspected, however, the serum lactate test—which costs only \$20—is a necessary first step in ruling out the condition. Because the standard of care required lactic acidosis to have been part of the differential diagnosis on October 1, based on Ms. Makombe’s symptoms, it was a further breach of the standard of care for Boers not to have ordered a serum lactate test at that time.

### **3. Lactic Acidosis Also Should Have Been Suspected on October 3**

Even more evidence supports a conclusion that lactic acidosis should have been on the differential diagnosis on October 3, when Falk consulted with Dr. Katz. It is undisputed that Ms. Makombe complained of actual shortness of breath when she came into the clinic on that day, as Falk noted that symptom on the triage form, in addition to Ms. Makombe’s continued complaints of vomiting and decreased appetite, and new complaints of weakness and dizziness. All are signs and symptoms of lactic acidosis. For the same reasons that Ms. Makombe’s respiratory complaint on October 1 should have led to suspicion of lactic acidosis on that date, her complaint of actual shortness of breath on October 3, combined with the new complaints of weakness and dizziness, required a differential diagnosis of lactic acidosis on that day as well.

In addition, Dr. Katz on October 3 reviewed the results of Ms. Makombe’s abdominal ultrasound, which showed that she had gallstones. The government asserts that these results “completely support[.]” a differential diagnosis of gallbladder disease rather than lactic acidosis. (Def.’s Closing Argument 12/19/06.) In fact, both parties’ experts testified to the contrary. Dr. Rumans testified that based on the ultrasound, Dr. Katz should then have been able to rule out gallbladder disease as the cause of Ms. Makombe’s signs and symptoms, as the test showed gallstones but not cholecystitis, or gallbladder inflammation. Dr. Kessler agreed that it was “clear” on October 3 that Ms. Makombe did not have cholecystitis, and that her symptoms were not caused by gallstones. Both experts testified, moreover, that Ms. Makombe’s September 18 complaint of abdominal pain in the upper left quadrant, which had prompted Tornabene to order the ultrasound,

was inconsistent with gallbladder disease, which presents with pain in the upper right quadrant. Finally, as Dr. Rumans pointed out, cholecystitis would not explain Ms. Makombe's complaint of shortness of breath in any event.<sup>36</sup>

Ms. Makombe did have a blood oxygenation rate of 98% on October 3, according to the pulse oximeter test ordered by Dr. Katz. This result does not, however, provide any reason for Dr. Katz to have discounted Ms. Makombe's complaint of shortness of breath. Dr. Rumans explained that the level of oxygen in the blood "reveals nothing" about acidosis, and that if a patient complains about shortness of breath but is oxygenating normally, then in fact further investigation is required. Similarly, Dr. Kessler admitted that oxygenation rate is unrelated to lactic acidosis, and that patients with Zerit-related lactic acidosis have normal levels of oxygen in both blood and tissue. These opinions are borne out by the fact that upon admission to Weiss on October 22, 2001, when it is undisputed that Ms. Makombe's lactic acidosis was irreversible, her oxygenation rate was 100%.

The government points out that the ultrasound results also showed that Ms. Makombe's liver was normal when the test was conducted on October 1. The government contrasts that result with the autopsy report, which showed that Ms. Makombe had a large, fatty liver when she died on October 24, 2001. (000144-000152.) According to the CDC Guidelines, a fatty liver is a sign and symptom of lactic acidosis; as that sign and symptom was absent on October 1, the government contends, there was no reason to suspect lactic acidosis. (Def.'s Closing Argument 12/19/06.) No witness testified, however, that a large or fatty liver is the most dispositive sign of lactic acidosis. Indeed, of all the signs and symptoms of lactic acidosis, shortness of breath appears to be the one symptom that the medical experts in this case agreed is a tell-tale sign. Moreover, there is no

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<sup>36</sup> In closing arguments, the government acknowledged that gallbladder disease does not itself cause shortness of breath, but asserted that "it does cause pain and pain causes a patient to feel short of breath." (Def.'s Closing Argument 12/19/06.) Farrington did testify that the shortness of breath that is associated with lactic acidosis occurs as a result of abdominal pain, but this testimony conflicts with that of both Dr. Kessler and Dr. Rumans, who explained that lactic acidosis-related shortness of breath results from metabolic processes at the cellular level.

evidence that Dr. Katz suspected lactic acidosis on October 3 and then ruled it out after seeing a normal liver on the ultrasound; rather, she never considered it in the first place. Given the myriad of other symptoms of lactic acidosis that Ms. Makombe demonstrated on October 3, 2001, Dr. Katz should have suspected lactic acidosis based on the clinical presentation alone.

Plaintiff further argues that Dr. Katz breached the standard of care by failing to review Ms. Makombe's chart on October 3, or to personally examine her. Dr. Rumans indeed so testified, but the court need not decide the issue. Certainly, a cursory review of the chart would have shown the extended duration of Ms. Makombe's GI-related symptoms, as well as the fact that she had come in just two days before and complained of respiratory problems. The court also finds unsatisfactory Dr. Katz's explanation that she depended on the nurses to "tell [her] that they think something is different." Yet based only on Ms. Makombe's presenting symptoms on October 3, lactic acidosis should have been on a differential diagnosis, regardless of whether Dr. Katz examined Ms. Makombe or reviewed her records. Dr. Katz's failure to consider lactic acidosis and to order a serum lactate test thus constituted breaches of the standard of care.

#### **4. Lactic Acidosis Also Should Have Been Suspected on October 9**

Even if the standard of care did not require Clinic staff to have suspected lactic acidosis on October 1 and October 3, 2001, the condition clearly should have been part of a differential diagnosis on October 9. On that day, Ms. Makombe presented with two new symptoms: a twelve-pound weight loss since August 28, and, most significantly, pain in both legs and paresthesia. Both are signs and symptoms of lactic acidosis.

The government contends that it was reasonable for Tornabene, who examined Ms. Makombe on October 9 and reviewed her chart, to have maintained a working diagnosis of gallbladder disease, and to have concluded that Ms. Makombe's new symptoms of leg soreness and paresthesia were independent side effects of Zerit. (Def.'s Closing Argument 12/19/06.) As explained above, however, both Dr. Rumans and Dr. Kessler testified that gallbladder disease

should have been ruled out back on October 3; and in any event, gallbladder disease would not have explained Ms. Makombe's other symptoms, most notably her shortness of breath. While it is true that Zerit can cause a peripheral neuropathy independent of lactic acidosis, that was unlikely, as Dr. Rumans testified, in light of the combination of Ms. Makombe's symptoms and their progression since August. Indeed, Dr. Rumans testified, and Dr. Kessler agreed, that lactic acidosis would have provided a medically-desirable unifying diagnosis that could have explained all of Ms. Makombe's symptoms.<sup>37</sup>

By October 9, Ms. Makombe had demonstrated the full panoply of signs and symptoms of the disease: vomiting, nausea, lack of appetite, weight loss, abdominal pain, dizziness, weakness, shortness of breath, muscle pain, and paresthesia. Although she reported on October 9 that her eating had improved, Tornabene and Dr. Rumans both testified that the Ranitidine, which Dr. Katz had prescribed for GERD on October 3, may have been masking epigastric pain, thus allowing her to eat. Tornabene also testified that she reviewed the results of Ms. Makombe's liver enzyme tests that Boers had ordered on October 1, showing AST and ALT elevations outside the normal range. Although she characterized these results as "essentially normal" because patients with HIV often have elevated liver enzymes, the court agrees with Fields that the elevation, which is consistent with lactic acidosis, should have prompted further inquiry in light of all of Ms. Makombe's other signs and symptoms.

The government urges that these symptoms suggest lactic acidosis only in "hindsight," but

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<sup>37</sup> Even if Tornabene's diagnosis of a peripheral neuropathy secondary to Zerit was reasonable, that conclusion itself may have provided a reason to discontinue the Zerit on October 9. The package inserts for Zerit in 1999, 2000, and 2001 warn that if a patient develops symptoms of peripheral neuropathy, then "stavudine therapy should be interrupted." (PDRs, Pl.'s Ex. 50-52.) Dr. Rumans confirmed that warning. Dr. Kessler disagrees with the FDA-approved instructions, however, and because Dr. Rumans did not explicitly testify that the standard of care requires removal of Zerit when a patient presents with signs of a peripheral neuropathy, the court is not inclined to find that Tornabene breached the standard of care on that basis.

the court disagrees. These symptoms were all either physically present on October 9 or were indicated in Ms. Makombe's chart. Given that lactic acidosis was a well-known side effect of Zerit, that the package insert and the CDC Guidelines warned of the condition, that the condition was potentially fatal, that Ms. Makombe was particularly at risk, and that Ms. Makombe by October 9 had demonstrated nearly all the signs and symptoms of lactic acidosis, Tornabene did not need the benefit of hindsight to suspect lactic acidosis.

Finally, and perhaps most significantly, Ms. Makombe's signs and symptoms on October 22, 2001, when Tornabene did put lactic acidosis on the differential, were substantially the same as her signs and symptoms on October 1, October 3, and October 9. On October 22, Ms. Makombe came on her own to the Clinic, complaining of shortness of breath that felt "like suffocating," severe pain in her ankles and the soles of her feet, epigastric pain, no eating, vomiting, dizziness and weakness. She had reported these complaints before, to varying degrees. Falk also noted increased abdomen size on the triage form, but even that symptom was not new, as Falk had indicated on September 18 that Ms. Makombe's abdomen was large and firm. Indeed, the only new factor on October 22 was that Ms. Makombe, according to Tornabene who observed her from several feet away, "looked awful." Although the government argues that the fact that lactic acidosis was not suspected until Ms. Makombe "looked awful" simply proves how difficult the condition is to diagnose, the court is unconvinced that such a criterion should define the standard of care for diagnosing a potentially lethal side effect of medication. As noted, lactic acidosis is diagnosed according to the signs and symptoms demonstrated by the patient; and Ms. Makombe had demonstrated, by October 9, every one of the symptoms she presented with on October 22.

#### **5. The Court Need Not Consider Plaintiff's Other Theories of Liability**

Plaintiff also attributes other breaches of the standard of care to specific clinic personnel and to the structure of the Clinic itself. Specifically, Plaintiff contends that Falk should have known the signs and symptoms of lactic acidosis in 2001, and that lactic acidosis was a potential side effect

of Zerit; that Boers and Dr. Katz, as noted above, should have examined Ms. Makombe personally when consulted by Falk; and that the Clinic's structure allowed Falk to become a de facto care provider, a role beyond the scope of her practice as an RN. (Pl.'s Closing Argument 12/19/06.) Plaintiff also solicited a good deal of testimony regarding Dr. Katz's and Tornabene's responsibilities under the CA. The court has already addressed the fact that Boers and Dr. Katz did not examine Ms. Makombe, concluding that the issue need not be resolved here; similarly, the court need not determine Falk's responsibilities, nor whether the Clinic's structure was deficient, nor the scope of the CA.

The evidence does appear to support Plaintiff's view that the standard of care required Falk, as an RN, to know in 2001 that lactic acidosis was a side effect of Zerit, and to know the signs and symptoms of the condition; Fields so opined, and Farrington admitted as much on cross-examination. For purposes of establishing the government's liability in this case, however, this finding is unnecessary. First, the individual Clinic personnel are not separate defendants in this action. Second, in light of the court's conclusion that the signs and symptoms with which Ms. Makombe presented on October 1, October 3, and October 9, 2001 required a differential diagnosis of lactic acidosis, it does not matter which Clinic employee was responsible: if Falk's role was simply to report a patient's symptoms to a care provider, as the government maintains, but she did not fully convey Ms. Makombe's symptoms to Boers, Dr. Katz, and Tornabene, respectively, then those care providers were prevented from making an accurate assessment; and if Falk did accurately report those signs and symptoms, the responsibility for the missed diagnosis lies with the care providers. As Falk and the care providers are all employees of the United States for purposes of this action, the government is liable either way. For the same reasons, the court need not determine whether the Clinic's structure allowed Falk to practice beyond the scope of her role as an RN, or who bears the ultimate responsibility for Ms. Makombe's care and treatment under the CA.

Finally, the court need not address Plaintiff's argument that Tornabene, Ms. Makombe's primary care provider, may have been unaware in 2001 that lactic acidosis was a side effect of Zerit. Clinic Executive Director Nelson testified in her deposition that when she told Tornabene and Dr. Katz of this lawsuit, Tornabene replied that lactic acidosis was not a known side effect of Zerit. At trial, Nelson further recalled that Tornabene stated that lactic acidosis was not listed as a side effect of Ms. Makombe's "medications"; but Nelson was unsure whether Tornabene was referring specifically to the Zerit. Although Nelson's testimony may be viewed as inconsistent with Tornabene's assertion in her testimony that she had indeed known in 2001 that lactic acidosis was a side effect of Zerit, the court need not make a finding of fact on the issue: the court's conclusion that Clinic staff should have suspected lactic acidosis as early as October 1 was based, as explained above, on Ms. Makombe's presenting symptoms, and any particular care provider's knowledge of the side effects of Zerit was not necessary to that conclusion.

**6. The Court Does Not Give Greater Weight to the Testimony of the Government's Experts**

Finally, the court briefly addresses the government's argument that the testimony of Plaintiff's experts, particularly Dr. Rumans, is not entitled to as much weight as that of Dr. Kessler and Farrington. The government makes much of the fact that Dr. Kessler is well-known in the AIDS care field, whereas Dr. Katz and Tornabene had never heard of Dr. Rumans prior to this lawsuit, and that Dr. Rumans, unlike Dr. Kessler, does not attend AIDS conferences or belong to HIV or AIDS organizations. Certainly, Dr. Kessler's curriculum vitae is impressive. The court does not, however, accept the government's contention that Dr. Rumans is "not in Dr. Kessler's league." (Def.'s Closing Argument 12/19/06.) Significantly, the government identifies no specific aspect of Dr. Kessler's background or experience that would make his opinion more valuable as to the issue at the heart of this lawsuit, i.e., what the standard of care requires for diagnosing lactic acidosis secondary to Zerit. In the court's view, clinical experience working with AIDS patients is the most

relevant credential with respect to that issue, and Dr. Kessler appears to hold no advantage over Dr. Rumans in this regard: both maintain a part-time clinical practice in AIDS and HIV care, and Dr. Kessler in fact testified that his clinical work consists of supervising other care providers. Moreover, the court found Dr. Rumans an impressive witness, and his testimony coherent, concise and persuasive.

The government also contends that the court should give more weight to Farrington's testimony than to Fields' because the latter has not treated a patient with lactic acidosis, whereas Farrington "lives and breathes this stuff" and even had the condition himself. (Def.'s Closing Argument 12/19/06.) The court agrees that Farrington, having developed lactic acidosis as a side effect of diabetes medication, brings a unique perspective. The court found his testimony less helpful, however, particularly as he admitted to testifying in his deposition that the reason lactic acidosis was not required to be part of a differential diagnosis in this case before October 22, 2001 was that no warnings of lactic acidosis appeared on the package inserts for Zerit prior to 2002. In fact, as noted, the inserts prominently displayed "black box" warnings as early as 1999. In contrast, the court found Fields' testimony, like that of Dr. Rumans, to be clear, well-reasoned, and persuasive.

#### **B. Proximate Cause**

A plaintiff in a medical malpractice case establishes proximate cause by proving, generally through expert testimony, that the defendant's breach of the applicable standard of care more probably than not caused her injury. *Newell*, 125 Ill. App. 3d at 1092, 466 N.E.2d at 1088; see *Borowski v. Von Solbrig*, 60 Ill. 2d 418, 424, 328 N.E.2d 301, 305 (1975). As explained above, the court has concluded that Ms. Makombe's care providers breached the standard of care by failing to suspect and test for lactic acidosis on October 1, October 3, and October 9, 2001. According to Dr. Rumans, serum lactate and arterial blood gas tests would have indicated lactic acidosis as early as September 26, and the government presents no evidence to the contrary. It is undisputed that the

treatment for lactic acidosis secondary to Zerit is removal of the Zerit. Dr. Rumans further testified that had the Zerit been discontinued on October 1, October 3, or, significantly, even as late as October 9, 2001, Ms. Makombe would have made a full recovery. This opinion went unchallenged. The court concludes that Plaintiff has sufficiently proved that the above breaches of the standard of care proximately caused Ms. Makombe's death, and therefore finds the government liable on Plaintiff's claim for negligence and wrongful death.

## **II. Damages**

Mr. Kasongo and his children have suffered a devastating loss as the result of Ms. Makombe's untimely death. A monetary award, at best an imperfect remedy, is the only remedy available to this court. The court addresses separately the parties' arguments concerning wrongful death and survival damages.

### **A. Damages for Wrongful Death**

Under the Illinois wrongful death statute,

surviving heirs or next of kin . . . are entitled to recover for the pecuniary losses incurred as a result of the death, including money, benefits, goods, services, and society. Although those damages do not include grief or mental anguish resulting from the death, damages for loss of society are recoverable, which include the deprivation of love, companionship, and affection from the deceased person.

*Turner v. Williams*, 326 Ill. App. 3d 541, 548, 762 N.E.2d 70, 77 (2nd Dist. 2001). A surviving spouse's recovery for loss of consortium includes loss of society, companionship, and sexual relations. *Elliott v. Willis*, 92 Ill. 2d 530, 541, 442 N.E.2d 163, 168 (1982); Illinois Pattern Jury Instruction ("IPI") 31.04. In addition, Illinois law recognizes a surviving spouse's right to recover for the loss of the deceased's spouse contributions to child rearing and family decision-making, and assistance with household chores. *Wood v. Mobil Chem. Co.*, 50 Ill. App. 3d 465, 478, 365 N.E.2d 1087, 1096 (5th Dist. 1977); see IPI 32.03.

In a wrongful death action brought by a surviving spouse or for lineal next of kin, "the law presumes substantial pecuniary damages arising from the relationship alone[.]" *Dotson v. Sears*,

*Roebuck and Co.*, 157 Ill. App. 3d 1036, 1052, 510 N.E.2d 1208, 1218 (1st Dist. 1987). Illinois courts will instruct a jury that in determining such pecuniary loss and the weight given to this presumption, the jury can consider the decedent's companionship, guidance, advice, love and affection as they were likely to occur in the future. *Id.*; see generally IPI 31.01 *et seq.* It is also appropriate to consider evidence of the quality of the relationships between the decedent and his or her children. See *Dotson*, 147 Ill. App. 3d at 1052, 510 N.E.2d at 1218; *Cooper v. Chicago Transit Auth.*, 153 Ill. App. 3d 511, 520, 505 N.E.2d 1239, 1244 (1st Dist. 1987).

Plaintiff, citing the hardships faced by Ms. Makombe's family in Africa and in their journey to the United States, the young ages of her children at the time of her death—Moises was ten, Ange was seven, and Sara was five—and the closeness of their relationships with their mother, seeks an award of damages of \$4 million for wrongful death, primarily on grounds of loss of society. (Pl.'s Br., at 10.) The government, contending that Ms. Makombe was in poor physical and mental condition due to AIDS, PTSD, and depression, and that her life expectancy was "limited," suggests a total award of \$700,000.<sup>38</sup> (Def.'s Br., at 3.) The government does not indicate whether this suggested award encompasses both wrongful death and survival damages.

In *Jutzi-Johnson v. United States*, 263 F.3d 753, 758-59 (7th Cir. 2001), the Seventh Circuit in a Federal Tort Claims Act case concluded that courts should examine damages awards in similar cases, when determining the value of pain and suffering, to limit "arbitrary variance in awards." Although *Jutzi-Johnson* involved the use of comparable verdicts in determining an appropriate award for pain and suffering, the loss of society, as claimed here, is analogous due to its intangible nature.

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<sup>38</sup> The court notes that Illinois in 2005 enacted a statutory cap on non-economic damages in medical malpractice cases, limiting such damages to \$1 million if awarded against a hospital and \$500,000 if awarded against a physician. See 735 ILCS 5/2-1706.5 (effective August 25, 2005). Because the cap only applies to causes of action accruing on or after the effective date of the legislation, the court need not determine its effect here.

More recently, a bill pending in the Illinois legislature would allow a jury in a wrongful death case to award damages for grief, sorrow, and mental suffering to the surviving spouse and next of kin of the deceased; it also does not apply retroactively. See HB 1798, 95th Gen. Assem. (Ill.).

The court therefore reviews awards for wrongful death in cases similar to this one. To this end, the parties have submitted a number of verdicts they consider comparable.<sup>39</sup> Before discussing these awards, however, the court will address the government's arguments concerning Ms. Makombe's health prior to her death, and her life expectancy.

### **1. Ms. Makombe's Health and Life Expectancy**

The government asserts that Ms. Makombe was "in poor physical and mental condition because of her ongoing AIDS diagnosis and because of the trauma she experienced before arriving in the United States." (Def.'s Br., at 2.) The government points to her "diagnoses" of PTSD and depression, for which the government contends Ms. Makombe refused treatment, and to the "Report of Incapacity" that Tornabene completed for the Illinois Department of Human Services on April 17, 2001, which indicated reduced capacities in many physical and mental activities. (*Id.*) The government also contends that according to the expert reports submitted by Dr. Rumans and Dr. Novak, Ms. Makombe had only a ten-year life expectancy.

The court finds these arguments unpersuasive, for several reasons. First, it is not at all clear that Ms. Makombe was in "poor physical and mental condition" before she developed lactic acidosis. Although she had AIDS, it is undisputed that she had never had an opportunistic AIDS-related infection, and that she demonstrated an excellent response to HAART treatment. Indeed, after she began HAART in September 2000, her T-cell count increased from 48 in July 2000 to 112 in January 2001, and her viral load plunged from more than 90,000 to a level considered undetectable. Falk testified that Ms. Makombe reported only "run-of-the-mill" complaints until April 2001. Ms. Makombe did experience episodic dizziness and blackouts in April and May 2001, which Tornabene suspected

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<sup>39</sup> The government also submits several published settlement reports. *Jutzi-Johnson* did not explicitly call for courts to consider settlement amounts when determining an award of damages, and any such comparison is less useful than consideration of verdicts. Parties settle claims to avoid the uncertainty and expense of trial; and the amount settled for may bear little relation to the amount a jury might award upon finding a defendant liable.

may have been due to a mild HIV-related encephalopathy; because Ms. Makombe could not tolerate the MRI, however, that condition was never actually diagnosed, and in any event, by September 18, 2001, Ms. Makombe reported that her blackouts had ceased.

Nor does the evidence entirely support the government's assertion that Ms. Makombe was "diagnose[d]" with PTSD and depression (though such an assessment does not appear unreasonable in light of Ms. Makombe's horrific experience in the Congo), or that Ms. Makombe "refused" mental health treatment. She did decline Tornabene's referrals to psychiatrists at the Kolver center; yet she appeared at many Clinic appointments with her case worker Martine, whom Falk described as Ms. Makombe's "mental health case worker from [Heartland] Refugee Mental Health." (000039.) The fact that prayer appeared to be Ms. Makombe's "treatment of choice," as Tornabene indicated in her notes, is not obviously relevant to her life expectancy, in any event.

The government is on firmer evidentiary ground in citing the state disability form completed by Tornabene. Physically, Tornabene indicated that Ms. Makombe had a 20-50% reduced capacity in the activities of walking, bending, turning, standing, climbing and stooping; an up-to-20% reduced capacity in sitting, speaking, pushing, travel, and grasping manipulations; and a lifting limit of ten pounds. Mentally, Tornabene indicated a greater than 50% reduced capacity in activities of daily living, in social functioning, and in concentration, persistence, and pace. (000167.) The relevance of these findings to the compensable elements of pecuniary loss allowed by Illinois law for a wrongful death claim is not immediately apparent, however. The government cites no authority in its damages brief, and fails to provide its reasoning in citing the disability report. The court notes that Illinois jury instructions for wrongful death allow a jury to consider the "health" of an adult decedent when considering the pecuniary loss for which a surviving spouse and next of kin are entitled to compensation, see IPI 31.04; but the government fails to explain how the limitations indicated on the disability form inhibited Ms. Makombe's relationships with Mr. Kasongo and her children, or her ability to provide guidance, affection, and companionship. These are the elements of "loss of

society” for which Plaintiff seeks compensation; Ms. Makombe’s lifting or stooping abilities are not dispositive factors. The court finds much more relevant Mr. Kasongo’s testimony that his wife was the “foundation of our household,” his “advisor,” and the children’s “guide” who provided “all the affection” his children had.<sup>40</sup>

The government’s argument that Ms. Makombe had a limited life expectancy might be relevant to compensable damages, as that would impact the amount of time during which her death deprived her husband and children of her guidance, companionship, love, and affection. The court is not persuaded, however, that the evidence supports the government’s assertion that Ms. Makombe had only a ten-year life expectancy. Dr. Novak, who testified on behalf of Plaintiff regarding Ms. Makombe’s life expectancy, opined that it was more probable than not that she would have a normal life expectancy, which, according to tables from the federal government, would be eighty-one years; she was thirty-nine when she died. Dr. Rumans testified that a ten-year survival estimate was “reasonable,” but Ms. Makombe could live for fifteen or twenty years, and with further advancements in HIV treatment, “maybe longer.” Although the government asks the court to disregard Dr. Rumans’ and Dr. Novak’s opinions at trial as contradicting their expert reports, (Def.’s Br., at 2), the court is not inclined to do so.

In his expert report, Dr. Novak opined that a “conservative estimate” of Ms. Makombe’s life expectancy would be “at least a 50% likelihood of surviving 10 years, had she continued on potent antiretroviral therapy,” and assuming no new advancements in treatment of HIV within that ten-year period. (Novak Report, Pl.’s Ex. 44.) The court does not share the government’s view that this statement necessarily conflicts with Dr. Novak’s testimony at trial. A statement that an individual has

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<sup>40</sup> Limitations on Ms. Makombe’s physical activities would be relevant if Plaintiff were arguing for “loss of services” damages. There was little evidence at trial of Ms. Makombe’s contributions to household chores and activities, apart from the fact that Falk gave Ms. Makombe a referral for home help on October 3, 2001, when Ms. Makombe complained of new symptoms of weakness and dizziness. In any event, Plaintiff couches the request for damages in terms of loss of society, rather than loss of services.

“at least a 50% likelihood of surviving 10 years” does not mean that it is more probably true than not that she will live *only* ten years. Nor does the court find Dr. Rumans’ testimony in conflict with his report. Although he stated in his report that Ms. Makombe could have lived “as long as 10 years,” he added that this was “barring any other advancements in this area of medicine.” At trial, he explained that he had also given this opinion in his deposition in 2002, but that since then, newer classes of HIV drugs had appeared and improvements had been made in existing HAART medications. It was in light of these advances that he opined that Ms. Makombe could live for fifteen or twenty years, and possibly even longer with further advancements.

Although Dr. Rumans’ and Dr. Novak’s testimony was not identical to the opinions expressed in their expert reports, the court finds their explanations of the discrepancies satisfactory. The court also overrules the government’s belated objection, raised for the first time in its damages brief, that Dr. Rumans’ and Dr. Novak’s opinions regarding life expectancy were not fully disclosed in their expert reports, in violation of the Federal Rules of Civil Procedure. (Def.’s Br., at 2 n.1.) See FED. R. CIV. P. 26(a)(2)(B) (expert reports “shall contain a complete statement of all opinions to be expressed”) & 37(c)(1) (party not permitted to use evidence that the party failed to disclose as required by Rule 26(a)). The government failed to object to Dr. Rumans’ or Dr. Novak’s testimony on this basis at trial, thus waiving the objection. Moreover, as explained above, the court does not view the opinions expressed at trial as necessarily inconsistent with what was presented in their reports.

Significantly, the government presented no evidence of its own regarding Ms. Makombe’s life expectancy, and on cross-examination of Drs. Rumans and Novak, failed to mount any credible challenge to their opinions. Indeed, the court found both experts’ testimony on the issue adequately supported. Dr. Novak noted a study, encompassing some 9,000 patients, the results of which indicated that an HIV patient who demonstrated Ms. Makombe’s T-cell count and viral load, in response to HAART, would have a 97.5% chance of surviving the full three-and-a-half-year period

that formed the parameters of the study. In formulating an opinion beyond that time, he relied on his own experience with HAART: he testified that he had seen HIV transformed from a “death sentence” to a chronic disease like diabetes, one that “can be managed for an indefinite period of time.” He noted that in addition to Ms. Makombe’s positive lab results, she had been adherent to the HAART regimen, had never experienced an AIDS-related illness, and had gained weight and reported more energy. Dr. Rumans similarly noted Ms. Makombe’s CD4 count, reduction in viral load, compliance with medications, and lack of a history of opportunistic infections—all of which “bode well for the future.” He also explained, as noted, that new HIV drugs had appeared since 2002; he specifically identified protease inhibitors, a new class of medications that Ms. Makombe could have taken as part of the HAART regimen, had the Zerit been discontinued. Particularly persuasive was Dr. Rumans’ testimony that his HIV and AIDS patients were now complaining of conditions that “everyone else gets,” including heart disease and high cholesterol.

In light of the testimony of Plaintiff’s experts, and the fact that the government offered no evidence of its own regarding Ms. Makombe’s life expectancy, the court is unwilling to accept the government’s assertion that Ms. Makombe had only a ten-year life expectancy.

## **2. Comparable Verdicts**

The government cites five recent Illinois jury awards, ranging from \$300,000 to \$2.5 million, that include wrongful death damages.<sup>41</sup> First, in *Wertepny v. Louvain* (tried 2006), a Cook County jury awarded \$350,000 to the estate of a patient who died as a result of his psychiatrist’s failure to diagnose his (unidentified) mental illness. 2006 WL 2189691. The award consisted of \$300,000 in wrongful death damages for loss of society to the decedent’s wife and daughter, and \$50,000 for

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<sup>41</sup> *Hernandez v. Saint Catherine Laboure Clinic* (tried 2006), a case cited by the government involving a stillborn infant, resulted in settlement of \$500,000, not, as the government asserts, a jury award. 2006 WL 2547925.

Two of the wrongful death awards cited by the government are accompanied by survival damages, which the court discusses below.

pain and suffering as survival damages. The report offers no information regarding the ages, backgrounds, and relationships of the family members, nor the nature of the patient's illness.

In *Lemons v. Dave* (tried 2001), a Madison County jury awarded total damages of \$470,000 for a failure to diagnose bladder cancer. 2001 WL 34062364. The 58-year-old decedent left a husband and three adult children. The husband received \$150,000 for loss of society; the remainder of the award consisted of survival damages and medical expenses. Both the decedent and her children were significantly older than Ms. Makombe and her children, however, and the verdict report provides no information regarding the nature of their relationships. Moreover, the plaintiff sued under a "medical lost chance" theory, arguing that had cancer been diagnosed, the decedent may have survived or her life may have been prolonged; here, in contrast, it is undisputed that Ms. Makombe would have recovered had her lactic acidosis been timely diagnosed.

In *Davenport v. Provena Hospitals* (tried 2005), a 66-year-old mother of three adult children died from an aortic aneurysm, after doctors at the defendant hospital's emergency room sent her home with a diagnosis of neck muscle pain. 2005 WL 1491201. A Kane county jury awarded \$1.1 million, entirely for loss of society to the decedent's husband of 50 years, who was confined to a wheelchair with back problems and who claimed that his wife had assisted him with his disability and with household functions. The plaintiff had sought \$2 million, based on a life expectancy of 15 years and a 20% chance of the decedent dying in surgery had her condition been diagnosed; the defendant claimed she had a life expectancy of only five years and a 60% to 70% chance of dying on the operating table.

Again, however, the decedent was significantly older than Ms. Makombe, and did not leave young children. The fact that the award was slightly over half of what the plaintiff sought suggests that the jury found the decedent's life expectancy to be shorter than 15 years, and/or that she had at least some significant chance of dying in surgery, had she been properly diagnosed. As noted, neither circumstance is present here: Ms. Makombe's life expectancy was not as limited as the

government urges, and she would very likely have recovered from lactic acidosis, had the diagnosis been timely. The court also notes that the jury's award of \$1.1 million in *Davenport* is actually higher than the government's suggested figure of \$700,000 here.

The government also cites *Carranza v. Sherman Hospital* (tried 2006), a wrongful death case in which emergency room doctors failed to diagnose a heart defect in a 16-day-old infant. 2006 WL 3071790. The case resulted in a jury verdict of \$2.25 million for the parents' future loss of society; the plaintiff, who sought an award between that amount and \$2.5 million, had argued that the parents' life expectancy, combined, was 100 years. The court notes that this award would support the government's position here only if one were to consider the value of the loss of society to a parent, resulting from the death of a child, as necessarily greater than the value of the loss of society to a child, resulting from the death of a parent. The court is unwilling to accept that proposition, and indeed is inclined to take the opposite view.<sup>42</sup>

Defendant's citation to the verdict in *Frazier v. Nash* (tried 2003) is more relevant, although it suggests an award much higher than what the government argues for here. 2003 WL 23415183. There, the decedent, a single mother to twin daughters, was 47 years old when she died of a pulmonary embolism, following her discharge from the hospital where the defendant physician had performed unrelated thyroid surgery. The plaintiff claimed that the surgeon should have diagnosed the embolism when the decedent, before being discharged, had developed symptoms including fever and trouble breathing. The jury agreed and awarded \$2.5 million for loss of companionship and guidance to the decedent's daughters, who were in their mid-twenties at the time of the trial, five years after their mother's death. The plaintiff further claimed that the girls had a loving relationship with their mother, who had worked six days a week while raising them alone.

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<sup>42</sup> It is also worth noting that under the rationale of *Carranza*, in which the number of years that the survivors are deprived of the decedent's society is considered in the aggregate, the comparison does not favor the government: including Mr. Kasongo, Moises, Ange, and Sara, the total number of years in which the family will live without Ms. Makombe is significant.

Plaintiff also cites *Frazier*, and although the facts are not identical, the case does suggest that a similar award might be appropriate here. The fact that the decedent in that case was a single mother who raised her children alone might suggest a greater deprivation of society to the children than where, as here, one parent has survived. On the other hand, Ms. Makombe had three children, all of whom were much younger at the time of her death than the twins in *Frazier*, and given the hardships that the family endured in Africa and the fact that they had just emigrated to an unfamiliar country, the children's need for their mother and their consequent loss as a result of her death, is at least as palpable here. And Ms. Makombe's husband is himself a Plaintiff who has suffered a significant loss of his own.

In addition to *Frazier*, Plaintiff cites to eighteen jury verdicts in wrongful death cases involving female decedents of Ms. Makombe's age. Many of these are less helpful, as they involve situations in which the decedent left only adult children, or the verdict reports do not reveal the relationships of the survivors to the deceased or the portion of the awards designated as damages for loss of society.<sup>43</sup> The court finds two of these cases relevant, however. In *Madalinski v. St. Alexis Medical Center* (tried 2004), a 36-year-old mother died of an abdominal hemorrhage the day after giving birth, leaving a husband, a three-year-old, and the newborn. 2004 WL 3418197. The plaintiff, her husband, contended that the defendant hospital and physicians failed properly to monitor her condition and appreciate her complaints of pain. A Cook County jury awarded \$12.4 million, including \$4.5 million for loss of society; it is unclear how the \$4.5 million was allocated between the husband and the children.

The 40-year-old mother in *Olson v. Harpenau* (tried 2004 in Wisconsin state court) also left

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<sup>43</sup> The highest award cited by Plaintiff, more than \$13 million for loss of society to a decedent's husband and daughter, occurred in a malpractice case where the Illinois Appellate Court reversed the judgment and remanded for a new trial, including on the issue of damages. *Spyrka v. County of Cook*, 366 Ill. App. 3d 156, 171, 851 N.E.2d 800, 813 (1st Dist. 2006) (noting that the trial court had erroneously allowed the plaintiff to ask the jury to consider loss of society to the daughter in terms of her own life expectancy, rather than the decedent's).

a husband and two children, aged five and seven. 2004 WL 2843214. She complained of chest pains to her family physician several times in a two-month period, but was never given a cardiac workup and died from a heart attack. The jury valued the loss of society and companionship claims of the survivors at \$1.5 million, as part of a total award of more than \$2.5 million that the trial court reduced due to a Wisconsin statutory cap on damages.

As noted, Ms. Makombe died at the age of thirty-nine, leaving behind a husband, to whom she had been married for fourteen years, and three children aged five, seven, and ten. Although the above cases bear only a general similarity to this one, *Madalinski* and *Harpenau* suggest a range of \$1.5 million to \$4.5 million as an award for loss of society where a woman of Ms. Makombe's age leaves behind a husband and at least two minor children; and *Frazier's* award of \$2.5 million—\$1.25 million for each adult child—took into account the nature of the decedent's relationship with her children. In light of these awards, Plaintiff's request of \$4 million for loss of society is not unreasonable, and the government's suggestion of \$700,000 clearly is low.

In the court's view, the facts justify an award close to what Plaintiff seeks. Ms. Makombe, her husband, and her children endured extreme hardships in their country of origin. Mr. Kasongo and his wife kept their family intact through two wars, in a struggle to survive that took them from the Congo, to Rwanda, to Cameroon, and finally to Chicago. Having recently arrived in a strange country, and at young ages, the children needed their mother's guidance and support, and that need continues. Mr. Kasongo's testimony, in which he described his daughter asking him in the middle of the night to open her door because her mother might be waiting outside, and of his struggles to keep Moises from going down the wrong path in the wake of his mother's death, was deeply affecting. For his part, Mr. Kasongo has lost, among other things, his wife's assistance with raising their children, including her advice and participation in family decision-making.

In light of comparable jury verdicts and the circumstances of this case, the court awards Moises, Ange, and Sara Kasongo \$1 million each for loss of society, and Innocent Kasongo

\$500,000 for loss of society and consortium, for total wrongful death damages in the amount of \$3.5 million.

### **B. Survival Damages**

The Illinois survival statute does not create a statutory cause of action, but instead “allows a representative of the decedent to maintain those statutory or common law actions which had already accrued to the decedent before he died.” *Nat. Bank of Bloomington v. Norfolk & W. Ry. Co.*, 73 Ill. 2d 160, 172, 383 N.E.2d 919, 923 (1978); *Myers v. Heritage Enters., Inc.*, 332 Ill. App. 3d 514, 516, 773 N.E.2d 767, 769 (4th Dist. 2002). Unlike damages for wrongful death, which address the injury suffered by the deceased’s next of kin due to the loss of the deceased, a survival action allows for the recovery of damages for injuries personally sustained by the deceased up to the time of death. *Wyness v. Armstrong World Indus. Inc.*, 131 Ill. 2d 403, 410, 546 N.E.2d 568, 571 (1989) (citing *Murphy v. Martin Oil Co.*, 56 Ill. 2d 423, 308 N.E.2d 583 (1974)). As explained above, Plaintiff has established the Clinic’s negligence in failing to diagnose Ms. Makombe’s lactic acidosis on October 1, October 3, or October 9, 2001, and that that negligence proximately caused her death on October 24, 2001. The survival statute thus allows Plaintiff, as Ms. Makombe’s representative, to recover those compensatory damages to which she would have been entitled, had she been able to prosecute the negligence claim. See *Patch v. Glover*, 248 Ill. App.3d 562, 573, 618 N.E.2d 583, 591 (1st Dist.1993) (citation omitted).

Those damages include conscious pain and suffering prior to death, see *Murphy*, 56 Ill. 2d at 431, 308 N.E.2d at 587, however briefly experienced, see *Glover v. City of Chicago*, 106 Ill. App. 3d 1066, 1072, 436 N.E.2d 623, 628 (1st Dist. 1982). According to Illinois pattern jury instructions, survival damages are ascertained by taking into consideration the nature, extent, and duration of the injury. See IPI 31.10. Illinois courts do not, however, require evidence such as medical testimony to establish conscious pain and suffering; rather, lay testimony describing Ms. Makombe’s actions prior to her death, coupled with evidence concerning her injury, is sufficient to support a recovery.

See *Cretton v. Protestant Memorial Med. Ctr., Inc.*, 371 Ill. App.3d 841, 846-47, 864 N.E.2d 288, 299 (5th Dist. 2007) (citations omitted).

Plaintiff seeks \$2 million in survival damages for Ms. Makombe's pain and suffering. The government, as noted, suggests a total award of \$700,000, without differentiating between wrongful death and survival damages. Indeed, the government makes no explicit argument with regard to survival damages. As noted, however, two of the government's cited jury verdicts include survival damages as well as wrongful death damages, and the government also cites to one verdict consisting entirely of survival damages; the court considers these along with Plaintiff's submissions.

The jury in *Wertepny*, as noted above, awarded \$50,000 for pain and suffering as survival damages to the estate of a patient who died as a result of a failure to diagnose mental illness. 2006 WL 2189691. The case is unhelpful, as no information is available concerning the nature of the illness or the extent and duration of the pain suffered. In *Lemons*, also cited by the government and discussed above, the estate received \$250,000 for pain and suffering in survival damages. 2001 WL 34062364. As explained, the court finds any analogy to the present case wanting, given that *Lemons* was a "medical lost chance" case.

In *Recinto v. St. Joseph's Hospital* (tried 2003), the only other case cited by the government where a jury awarded survival damages, the plaintiff's decedent, a 69-year-old man who was a diabetic and an asthmatic, was released from the defendant hospital where he had received treatment for pneumonia. 2003 WL 23169388 (Nat. JVRA); 2003 WL 22170401 (VerdictSearch). He was then monitored at home by a visiting nurse; three days later, he was rushed to the emergency room, where he died of multi-system organ failure and lack of oxygen. The plaintiff contended that the nurse failed to recognize and relate to the defendant physician the severity of the decedent's breathing problems and the significance of his high blood sugar count, despite the impassioned pleas of family members who eventually called paramedics on their own. Although the jury found the hospital and physician not liable, the jury did find the nurse liable and awarded \$2

million in survival damages for pain and suffering. The decedent was conscious while suffering organ failure, and a medical expert testified as to the pain he suffered over the course of twenty-four hours before he expired.

The government's purpose in drawing this case to the court's attention is not evident; presumably, it is to illustrate that Ms. Makombe's conscious pain and suffering was not so extreme. It is not clear that this is so, however, and in any event, the duration of Ms. Makombe's pain was much longer than the twenty-four hours in *Recinto*. As Plaintiff points out, triage forms and treatment notes beginning on October 1, 2001, which the court has ascertained as the date Ms. Makombe's care providers first breached the standard of care by failing to have lactic acidosis on a differential diagnosis, show that she experienced significant pain from that point up to and through her admission to Weiss on October 22. On October 1, she reported abdominal pain, vomiting, and shortness of breath; the fact that she came in as a walk-in patient on that day, without an appointment, suggests that her discomfort was not minimal. Similarly, she came in again without an appointment on October 3, with additional complaints of weakness and dizziness. By October 9, she was experiencing pain "deep inside" both legs, and numbness. By October 22, her shortness of breath had progressed to the point where she felt that she was "suffocating," the pain in her feet and ankles was "severe," and she reported "burning" epigastric pain. When sent for further testing at Weiss Hospital, she expressed her fear that she would "die in the street" if she did not go immediately to the emergency room. Ms. Makombe was obviously in great pain on October 22, and that pain could only have increased as her body continued to fail her. Finally, although there was no testimony regarding Ms. Makombe's level of consciousness in the ICU at Weiss, the court notes that the autopsy report, summarizing her treatment at Weiss, indicates that she "was responsive to pain and voice" on October 23, before a paralytic agent was administered along with powerful sedatives. (000148.)

Plaintiff supports the request of \$2 million with verdicts in which juries have awarded up to

\$5 million in survival damages for pain and suffering. In *Madalinski*, discussed above, the jury awarded that amount based on just one hour of pain and suffering; the decedent complained of severe abdominal pain and right shoulder pain during that time, and she had two seizures before losing consciousness. 2004 WL 1857807. In *Christopher v. Rumsey* (tried 2002), a Cook County jury awarded \$2.5 million in survival damages where the defendant physician punctured the bladder of a 49-year-old patient during a tubal ligation procedure. She died nine days later of necrotizing fasciitis, which is known as the “flesh-eating” bacteria. 2002 WL 32374371; see also Access Plus Jury Verdict, No. 99L-2579. Of the award, \$500,000 was allocated to conscious pain and suffering; the remaining \$2 million was for disability and disfigurement, as treatment for the necrotizing fasciitis included removal of much of the patient’s external genitalia. In *Roth v. McTabi* (tried 2003), a Du Page County jury awarded \$200,000 for conscious pain and suffering where the decedent died of septic shock after arriving at the hospital complaining of fever, chills, vomiting, weight loss and appetite loss. 2003 WL 25540022; see also Access Plus Jury Verdict, No. 99L-1267. Physicians, mistakenly diagnosing alcohol withdrawal, gave her Valium; she lost consciousness the following morning and never regained it before her death.

The above awards show a significant variance in survival damages for pain and suffering. *Roth* and *Christopher* suggest that the award increases with the length of time that pain is suffered; yet the juries in *Recinto* and *Madalinski* awarded substantially higher damages—\$2 million and \$5 million, respectively—for much shorter durations, possibly because the juries heard explicit evidence of the decedent’s conscious pain. Although such explicit testimony is absent here, the court takes into account the long duration of Ms. Makombe’s complaints of pain, the escalation of that pain to the point where she feared she would “die on the street,” and the fact that she remained responsive to pain almost up to the moment of her death. The court concludes that an award of \$1 million is appropriate, and thus awards that amount to Plaintiff’s estate as survival damages for Ms. Makombe’s conscious pain and suffering prior to her death.

**CONCLUSION**

This case has been an exceedingly challenging one, for many reasons. There can be no question that the mission of Heartland and the Clinic is an admirable one, and the court is no less certain that Ms. Makombe's care providers, in particular Falk and Tornabene, are dedicated professionals who desired nothing more than to give Ms. Makombe the best care possible. Indeed, Ms. Makombe's death is particularly tragic, occurring as it did at a time when, thanks to the efforts of Heartland and the Clinic, it appeared that the worst of her ordeals may have been behind her: the family had escaped the horrors of war to begin a new life in the United States; Ms. Makombe was receiving state-of-the art HIV treatment; and the treatment was working. That she died from the very medication that was helping her, at a time when her life was full of promise, is wrenching.

For the reasons explained above, the court finds in favor of Plaintiff and awards \$4.5 million in damages: \$1 million in survival damages to Ms. Makombe's estate for her conscious pain and suffering; \$500,000 to Innocent Kasongo for loss of society and consortium; and \$1 million for loss of society to each of Ms. Makombe's three children.

ENTER:



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REBECCA R. PALLMEYER  
United States District Judge

Dated: July 16, 2007