

STATE OF NEW YORK
SUPREME COURT COUNTY OF WESTCHESTER

MICHAEL & JOAN REDA, husband and wife,

Plaintiffs,

COMPLAINT

Index No.:

-vs-

Bar Taco Port Chester, LLC d/b/a "bartaco"

Defendant.

COMPLAINT FOR DAMAGES

Plaintiffs, MICHAEL and JOAN REDA, through the undersigned attorneys allege upon information and belief for their Complaint against the Defendant Bartaco Port Chester, LLC d/b/a "bartaco", as follows:

I.

THE PARTIES

1. The Plaintiffs MICHAEL and JOAN REDA (hereinafter "the Plaintiffs"), at all times relevant to this Complaint, are and were a citizens and residents of the City of Yonkers and County of Westchester, State of New York.

2. The Defendant, Bar Taco Port Chester, LLC d/b/a as "bartaco" ("bartaco") at all material times owned and operated the restaurant known as bartaco located at 1 Willet Avenue, Port Chester, New York 13165 (Defendant's restaurant"). The Defendant at all times material hereto was carrying on in its ordinary course of business of the company, was in the business of the manufacture, distribution, preparation, service and sale of food to its store customers at that location, and as such was doing business in Port Chester, New York.

II.

JURISDICTION AND VENUE

3. This court is vested with original jurisdiction over the Defendant because it was doing business within the State of New York, pursuant to New York CPRL §§ 301, *et seq.*

4. The venue of this action is proper in Westchester County, pursuant to New York CPLR § 503, as the events giving rise to the cause of action occurred in Port Chester, New York, in Westchester County

III.

STATEMENT OF FACTS

5. The Plaintiffs repeat and reallege all prior paragraphs as if fully set forth herein.

About Hepatitis A Virus

6. Exposure to hepatitis A virus (“HAV”) can cause an acute infection of the liver that is typically mild and resolves on its own.¹ The symptoms and duration of illness vary a great deal, with many persons showing no symptoms at all.² Fever and jaundice are two of the symptoms most commonly associated with HAV infection.³

7. Throughout history, hepatitis infections have plagued humans. The “earliest accounts of contagious jaundice are found in ancient China.”⁴

8. According to the CDC:

The first descriptions of hepatitis (epidemic jaundice) are generally attributed to Hippocrates. Outbreaks of jaundice, probably hepatitis A, were reported in the 17th and 18th centuries, particularly in association with military campaigns. Hepatitis A (formerly called infectious hepatitis) was first differentiated

¹ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” in Mandell, Douglas, & Bennett’s PRINCIPLES AND PRACTICE OF INFECTIOUS DISEASES, Fifth Edition, Chap. 161, pp. 1920-40 (2000); Mayo Clinic Staff, “Hepatitis A,” (last updated Sept 1, 2011). Articles available online at <http://www.mayoclinic.com/health/hepatitis-a/DS00397>.

² Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

³ Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

⁴ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

epidemiologically from hepatitis B, which has a long incubation period, in the 1940s. Development of serologic tests allowed definitive diagnosis of hepatitis B. In the 1970s, identification of the virus, and development of serologic tests helped differentiate hepatitis A from other types of non-B hepatitis.⁵

9. Until 2004, HAV was the most frequently reported type of hepatitis in the United States. In the pre-vaccine era, the primary methods used for preventing HAV infections were hygienic measures and passive protection with immune globulin (IG). Hepatitis A vaccines were licensed in 1995 and 1999. These vaccines provide long-term protection against HAV infection.⁶

10. Hepatitis A is the only common vaccine-preventable foodborne disease in the United States.⁷ This virus is one of five human hepatitis viruses that primarily infect the human liver and cause human illness.⁸ Unlike hepatitis B and C, hepatitis A does not develop into chronic hepatitis or cirrhosis, which are both potentially fatal conditions.⁹ Nonetheless, infection with the hepatitis A virus (HAV) can lead to acute liver failure and death.¹⁰

11. Hepatitis A is a communicable (or contagious) disease that often spreads from person to person.¹¹ Person-to-person transmission occurs via the “fecal-oral route,” while all other exposure is generally attributable to contaminated food or water.¹² Food-related outbreaks are usually associated with contamination of food during preparation by a HAV-infected food

⁵ CDC, “Hepatitis A,” in EPIDEMIOLOGY AND PREVENTION OF VACCINE-PREVENTABLE DISEASES (also known as “The Pink Book”), Atkinson W, Wolfe S, Hambrosky J, McIntyre L, editors, 12th edition. Chapter available online at <http://www.cdc.gov/vaccines/pubs/pinkbook/hepa.html>.

⁶ *Id.*

⁷ *Id.*; See also Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” Clinical Infectious Diseases, Vol. 38, 705-715 (March 1, 2004). Full text online at http://www.cdc.gov/hepatitis/PDFs/fiore_ha_transmitted_by_food.pdf.

⁸ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

⁹ *Id.*

¹⁰ Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

¹¹ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

¹² *Id.*; See also Jaykus Lee Ann, “Epidemiology and Detection as Options for Control of Viral and Parasitic Foodborne Disease,” Emerging Infectious Diseases, Vol. 3, No. 4, pp. 529-39 (October-December 1997). Full text of the article is available online at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2640072/pdf/9366607.pdf>

handler.¹³ The food handler is generally not ill because the peak time of infectivity—that is, when the most virus is present in the stool of an infected individual—occurs two weeks before illness begins.¹⁴

12. Fresh produce contaminated during cultivation, harvesting, processing, and distribution has also been a source of hepatitis A.¹⁵ In 1997, frozen strawberries were the source of a hepatitis A outbreak in five states.¹⁶ Six years later, in 2003, fresh green onions were identified as the source of a HAV outbreak traced to consumption of food at a Pennsylvania restaurant.¹⁷ Other fruits and vegetables, such as blueberries and lettuce, have also been associated with HAV outbreaks in the U.S. as well as in other developed countries.¹⁸ HAV is relatively stable and can survive for several hours on fingertips and hands and up to two months on dry surfaces.¹⁹ The virus can be inactivated by heating to 185°F (85°C) or higher for one minute, or disinfecting surfaces with a 1:100 dilution of household bleach in tap water.²⁰ HAV can still be spread from cooked food if it is contaminated after cooking.²¹

¹³ Fiore, Anthony, *supra* note 7; CDC, “Hepatitis A,” *supra* note 5; See also CDC, “Surveillance for Acute Viral Hepatitis – United States, 2007, Morbidity and Mortality Weekly Report, Surveillance Summaries, Vol. 58, No. SS03 (May 22, 2009) at <http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5803a1.htm>.

¹⁴ Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7.

¹⁵ *Id.*; See also, Wheeler, C, *et al.*, “An Outbreak of Hepatitis A Associated with Green Onions,” New England Journal of Medicine, Vol. 353, 890-97 (2005). Full text of article available at <http://www.nejm.org/doi/full/10.1056/NEJMoa050855>.

¹⁶ Hutin YJF, *et al.*, “A Multistate, Foodborne Outbreak of Hepatitis A,” New England Journal of Medicine, Vol. 340, pp. 595-602 (1999). Full text of article is online at <http://nejm.org/doi/full/10.1056/NEJM199902253400802>.

¹⁷ Wheeler, C, *et al.*, “An Outbreak of Hepatitis A Associated with Green Onions,” *supra* note 15.

¹⁸ Butot S, *et al.*, “Effects of Sanitation, Freezing and Frozen Storage on Enteric Viruses in Berries and Herbs,” Intentional Journal of Food Microbiology, Vol. 126, No. 4, pp. 233-246 (2003). Full text of article is available at http://www.prograd.uff.br/virologia/sites/default/files/bulot_et_al_2008_inactivation.pdf; Calder, L, *et al.*, An Outbreak of Hepatitis A Associated with Consumption of Raw Blueberries,” Epidemiology and Infection, Vol. 131, No. 1 745-51 (2003) at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2870016/pdf/12948375.pdf>.

¹⁹ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

²⁰ CDC, “Updated recommendations from Advisory Committee on Immunization Practices (ACIP) for use of hepatitis A vaccine in close contacts of newly arriving international adoptees,” Morbidity and Mortality Weekly Report, Vol. 58, No. 36, (Sept. 18, 2006), <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5836a4.htm>; Fiore, Anthony, *et al.*, Advisory Committee on Immunization Practices (ACIP), Prevention of Hepatitis-A Through Active or Passive Immunization: Recommendations, Morbidity & Mortality Weekly Review, Vol. 55, Report 407, (May

13. Although ingestion of contaminated food is a common means of spread for HAV, it may also be spread by household contact among families or roommates, sexual contact, or by direct inoculation from persons sharing illicit drugs.²² Children are often asymptomatic, or have unrecognized infections, and can pass the virus through ordinary play, unknown to their parents, who may later become infected from contact with their children.²³

14. Hepatitis A may cause no symptoms at all when it is contracted, especially in children.²⁴ Asymptomatic individuals will only know they were infected (and have become immune, given that you can only get hepatitis A once) by getting a blood test later in life.²⁵ Approximately 10 to 12 days after exposure, HAV is present in blood and is excreted via the biliary system into the feces.²⁶ Although the virus is present in the blood, its concentration is much higher in feces.²⁷ HAV excretion begins to decline at the onset of clinical illness, and decreases significantly by 7 to 10 days after onset of symptoms.²⁸ Most infected persons no longer excrete virus in the feces by the third week of illness. Children may excrete HAV longer than adults.²⁹

29, 2006) at <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5507a1.htm>; Todd, Ewan C.D., *et al.*, “Outbreaks Where Food Workers Have Been Implicated in the Spread of Foodborne Disease. Part 6. Transmission and Survival of Pathogens in the Food Processing and Preparation-environment,” *Journal of Food Protection*, Vol. 72, 202-19 (2009). Full text of the article is available online at

http://courses.washington.edu/eh451/articles/Todd_2009_food%20processing.pdf.

²¹ Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7.

²² *Id.*; *See also*, Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

²³ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Piazza, M, *et al.*, “Safety and Immunogenicity of Hepatitis A Vaccine in Infants: A Candidate for Inclusion in Childhood Vaccination Program,” Vol. 17, pp. 585-588 (1999). Abstract at <http://www.ncbi.nlm.nih.gov/pubmed/10075165>; Schiff, E.R., “Atypical Manifestations of hepatitis-A,” *Vaccine*, Vol. 10, Suppl. 1, pp. 18-20 (1992). Abstract at <http://www.ncbi.nlm.nih.gov/pubmed/1475999>.

²⁴ Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7

²⁵ Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

²⁶ CDC, “Hepatitis A,” *supra* note 5; Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1

²⁷ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1

²⁸ *Id.*

²⁹ *Id.*; *See also* Sagliocca, Luciano, *et al.*, “Efficacy of Hepatitis A Vaccine in Prevention of Secondary Hepatitis A Infection: A Randomized Trial,” *Lancet*, Vol. 353, 1136-39 (1999). Abstract at [http://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(98\)08139-2/abstract](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(98)08139-2/abstract).

15. Seventy percent of HAV infections in children younger than six years of age are asymptomatic; in older children and adults, infection tends to be symptomatic with more than 70% of those infected developing jaundice.³⁰ Symptoms typically begin about 28 days after contracting HAV, but can begin as early as 15 days or as late as 50 days after exposure.³¹ The symptoms include muscle aches, headache, anorexia (loss of appetite), abdominal discomfort, fever, and malaise.³²

16. After a few days of typical symptoms, jaundice (also termed “icterus”) sets in.³³ Jaundice is a yellowing of the skin, eyes, and mucous membranes that occurs because bile flows poorly through the liver and backs up into the blood.³⁴ The urine will also turn dark with bile and the stool light or clay-colored from lack of bile.³⁵ When jaundice sets in, initial symptoms such as fever and headache begin to subside.³⁶

17. In general, symptoms usually last less than two months, although 10% to 15% of symptomatic persons have prolonged or relapsing disease for up to 6 months.³⁷ It is not unusual, however, for blood tests to remain abnormal for six months or more.³⁸ The jaundice so commonly associated with HAV can also linger for a prolonged period in some infected persons,

³⁰ CDC, “Hepatitis A,” *supra* note 5.

³¹ *Id.*; See also Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7.

³² CDC, “Hepatitis A,” *supra* note 5; Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

³³ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

³⁴ Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

³⁵ CDC, “Hepatitis A,” *supra* note 5; Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

³⁶ Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

³⁷ Fiore, Anthony, *et al.*, Advisory Committee on Immunization Practices (ACIP), Prevention of Hepatitis-A Through Active or Passive Immunization: Recommendations,” *supra* note 20; Gilkson Miryam, *et al.*, “Relapsing Hepatitis A. Review of 14 cases and literature survey,” Medicine, Vol. 71, No. 1, 14-23 (Jan. 1992). Abstract of article online at <http://www.ncbi.nlm.nih.gov/pubmed/1312659>.

³⁸ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

sometimes as long as eight months or more.³⁹ Additionally, pruritus, or severe “itchiness” of the skin, can persist for several months after the onset of symptoms. These conditions are frequently accompanied by diarrhea, anorexia, and fatigue.⁴⁰

18. Relapse is possible with hepatitis A, typically within three months of the initial onset of symptoms.⁴¹ Although relapse is more common in children, it does occur with some regularity in adults.⁴² The vast majority of persons who are infected with hepatitis A fully recover, and do not develop chronic hepatitis.⁴³ Persons do not carry HAV long-term as with hepatitis B and C.⁴⁴

19. Fulminant hepatitis A, or acute liver failure, is a rare but devastating complication of HAV infection.⁴⁵ As many as 50% of individuals with acute liver failure may die or require emergency liver transplantation.⁴⁶ Elderly patients and patients with chronic liver disease are at higher risk for fulminant hepatitis A.⁴⁷ In parallel with a declining incidence of acute HAV infection in the general population, however, the incidence of fulminant HAV appears to be decreasing.⁴⁸

³⁹ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

⁴⁰ CDC, “Hepatitis A,” *supra* note 5; Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

⁴¹ Gilkson Miryam, *et al.*, “Relapsing Hepatitis A. Review of 14 cases and literature survey,” *supra* note 37.

⁴² Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Gilkson Miryam, *et al.*, “Relapsing Hepatitis A. Review of 14 cases and literature survey,” *supra* note 37.

⁴³ Mayo Clinic Staff, “Hepatitis A,” *supra* note 1.

⁴⁴ CDC Summary, “Disease Burden from Viral Hepatitis A, B and C in the United States, 2004-2009, at http://www.cdc.gov/hepatitis/pdfs/disease_burden.pdf; CDC, “Hepatitis A,” *supra* note 5.

⁴⁵ Detry, Oliver, *et al.*, “Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management,” *World Journal of Gastroenterology*, Vol. 12, No. 46 pp. 7405-7412 (Dec. 14, 2006). Full article is available online at <http://www.wjgnet.com/1007-9327/12/7405.pdf>.

⁴⁶ Taylor, Ryan, *et al.*, “Fulminant Hepatitis A Virus Infection in the United States: Incidence, Prognosis, and Outcomes,” *Hepatology*, Vol. 44, 1589-1597. Full text

http://deepblue.lib.umich.edu/bitstream/2027.42/55879/1/21349_ftp.pdf.

⁴⁷ *Id.*; See also Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1.

⁴⁸ Taylor, Ryan, *et al.*, “Fulminant Hepatitis A Virus Infection in the United States: Incidence, Prognosis, and Outcomes,” *supra* note 46.

20. HAV infects the liver's parenchymal cells (internal liver cells).⁴⁹ Once a cell has been penetrated by the viral particles, the hepatitis A releases its own toxins that cause, in essence, a hostile takeover of the host's cellular system.⁵⁰ The cell then produces new viral components that are released into the bile capillaries or tubes that run between the liver's parenchymal cells.⁵¹ This process results in the death of liver cells, called hepatic necrosis.⁵²

21. The fulminant form of hepatitis occurs when this necrotic process kills so many liver cells—upwards of three-quarters of the liver's total cell count—that the liver can no longer perform its job.⁵³ Aside from the loss of liver function, fulminant hepatic failure can lead to encephalopathy and cerebral edema.⁵⁴ Encephalopathy is a brain disorder that causes central nervous system depression and abnormal neuromuscular function.⁵⁵ Cerebral edema is a swelling of the brain that can result in dangerous intracranial pressure.⁵⁶ Intracranial hypertensions leading to a brain stem death and sepsis with multiple organ failure are the leading causes of death in individuals with fulminant hepatic failure.⁵⁷

⁴⁹ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45; Feinstone, Stephen and Gust, Ian, "Hepatitis A Virus," *supra* note 1.

⁵⁰ Feinstone, Stephen and Gust, Ian, "Hepatitis A Virus," *supra* note 1; Schiff, E.R., "Atypical Manifestations of hepatitis-A," *supra* note 23.

⁵¹ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45.

⁵² *Id.*; See also Taylor, Ryan, *et. al.*, "Fulminant Hepatitis A Virus Infection in the United States: Incidence, Prognosis, and Outcomes," *supra* note 46.

⁵³ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45; Taylor, Ryan, *et. al.*, "Fulminant Hepatitis A Virus Infection in the United States: Incidence, Prognosis, and Outcomes," *supra* note 46.

⁵⁴ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45.

⁵⁵ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45; Feinstone, Stephen and Gust, Ian, "Hepatitis A Virus," *supra* note 1.

⁵⁶ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45.

⁵⁷ Detry, Oliver, *et al.*, "Brain Edema and Intracranial Hypertension in Fulminant Hepatic Failure: Pathophysiology and Management," *supra* note 45; Taylor, Ryan, *et. al.*, "Fulminant Hepatitis A Virus Infection in the United States: Incidence, Prognosis, and Outcomes," *supra* note 46.

22. Hepatitis A is much more common in countries with underdeveloped sanitation systems and, thus, is a risk in most of the world.⁵⁸ An increased transmission rate is seen in all countries other than the United States, Canada, Japan, Australia, New Zealand, and the countries of Western Europe.⁵⁹ Nevertheless, infections continue to occur in the United States, where approximately one-third of the population has been previously infected with HAV.⁶⁰

23. Each year, approximately 30,000 to 50,000 cases of hepatitis A occur in the United States.⁶¹ Historically, acute hepatitis A rates have varied cyclically, with nationwide increases every 10 to 15 years.⁶² The national rate of HAV infections has declined steadily since the last peak in 1995.⁶³ Although the national incidence—1.0 case per 100,000 population—of hepatitis A was the lowest ever recorded in 2007, it is estimated that asymptomatic infections and underreporting kept the documented incidence-rate lower than it actually is. In fact, it is estimated that there were 25,000 new infections in 2007.⁶⁴

24. In 2007, the CDC reported a total of 2,979 acute symptomatic cases of HAV.⁶⁵ Of these, information about food and water exposure was known for 1,047 cases, leading to an

⁵⁸ Feinstone, Stephen and Gust, Ian, “Hepatitis A Virus,” *supra* note 1; Jaykus Lee Ann, “Epidemiology and Detection as Options for Control of Viral and Parasitic Foodborne Disease,” *supra* note 12.

⁵⁹ CDC, “Update: Prevention of Hepatitis A after Exposure to Hepatitis A Virus and in International Travelers, Updated ACIP Recommendations,” *Morbidity and Mortality Weekly Report*, Vol. 56, No. 41, pp. 1080-84 (Oct. 19, 2007), online at <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5641a3.htm>.

⁶⁰ CDC, “Surveillance for Acute Viral Hepatitis – United States 2007,” *supra* note 13; Fiore, Anthony, Division of Viral Hepatitis, CDC, “Hepatitis A Transmitted by Food,” *supra* note 7.

⁶¹ CDC, Summary, “Disease Burden from Viral Hepatitis A, B, and C in the United States,” *supra* note 44; CDC, “Hepatitis A,” *supra* note 5.

⁶² Hutin YJF, *et al.*, “A Multistate, Foodborne Outbreak of Hepatitis A,” *supra* note 16.

⁶³ CDC, Summary, “Disease Burden from Viral Hepatitis A, B, and C in the United States,” *supra* note 44; CDC, “Surveillance for Acute Viral Hepatitis – United States 2007,” *supra* note 13.

⁶⁴ CDC, “Surveillance for Acute Viral Hepatitis – United States 2007,” *supra* note 13; Schiff, E.R., “Atypical Manifestations of hepatitis-A,” *supra* note 23.

⁶⁵ CDC, “Surveillance for Acute Viral Hepatitis – United States 2007,” *supra* note 13.

estimate that 6.5% of all infections were caused by exposure to contaminated water or food.⁶⁶ In 2,500 of the cases, no known risk factor was identified.⁶⁷

The HAV Outbreak

25. In October 2017, the Westchester County Department of Health (“DOH”) became aware of at least four persons who contracted hepatitis A as early as September 2017, -the commonality among them being that they all ate at the Defendant’s restaurant located at 1 Willett Ave, Port Chester, New York 10573.

26. On October 25, 2017, the DOH announced, that customers who visited the Defendant’s restaurant between October 12, 2017 and October 23, 2017 were exposed to the Hepatitis A Virus (“HAV”) through a bartaco’s infected employee. The DOH announced that persons who had consumed food or drink at Defendant’s restaurant were accordingly at imminent risk of infection with Hepatitis. DOH Officials further urged that these persons contact their health care provider to receive medically necessary treatment, including prophylactic vaccination with either a hepatitis A vaccine or immune-globulin, along with for some blood tests and other diagnostic procedures.

27. The DOH further announced that patrons who dined at the Defendant’s restaurant as early as August 22, 2017, should contact their health care provider immediately if they experience symptoms, since such patrons were outside the two-week window for effective prophylactic treatment.

JOAN and MICHAEL REDA’s Hepatitis A Illnesses

28. The Plaintiffs JOAN and MICHAEL REDA are both fifty-one-year-old residents of Yonkers, New York.

⁶⁶ *Id.*

⁶⁷ *Id.*

29. On September 10, 2017, the Plaintiffs purchased and consumed food and drink at Defendant's restaurant known as "bartaco" located at 1 Willett Ave, Port Chester, New York 10573.

30. The Plaintiff MICHAEL REDA began experiencing the onset of symptoms on October 14, 2017. These included vomiting, diarrhea, stomach cramps, muscle aches, fatigue, headache, dark urine, light fecal matter, and jaundice.

31. On October 19 and 20, the Plaintiff MICHAEL REDA sought medical attention at Lawrence Hospital. A blood sample taken from the Plaintiff at the facility tested positive for hepatitis A.

32. The Plaintiff MICHAEL REDA sought additional medical treatment for his HAV at Cornell Medical Center on October 20 through 23.

33. The Plaintiff MICHAEL REDA attends regularly-scheduled follow-up appointments with his primary care physician, Veronique Brinson, MD, every two weeks for ongoing management of his continuing HAV symptoms.

34. The Plaintiff MICHAEL REDA was forced to stay home from work for nearly two months without pay, and has accumulated substantial medical expenses.

35. The Plaintiff JOAN REDA submitted a blood sample at CityMD Urgent Care on November 14, 2017 which tested positive for hepatitis A.

36. The Plaintiff JOAN REDA began experiencing symptom onset on October 30, 2017. Her symptoms included nausea, stomach cramps, muscle aches, fatigue, headache, jaundice, and dark urine.

37. The Plaintiff JOAN REDA received medical attention at Montefiore Medical Group for her illness from November 15, 2017 to the present.

38. The Plaintiff JOAN REDA was debilitated for nearly three weeks because of her illness. And she continues to struggle with her symptoms at work. Her substantial fatigue, nausea, and headaches (along with experiencing dark colored urine) significantly inhibited her ability to engage in regular activities such as walking, training, and driving.

39. The Plaintiffs were eventually contacted by Jenny Linus from the Westchester County Health Department, who interviewed the Plaintiffs regarding their diagnosis and illness.

IV.

FIRST CAUSE OF ACTION (Breach of Warranties)

40. The Plaintiffs repeat and reallege all prior paragraphs as if fully set forth herein.

41. At all material times, the Defendant was and is the owner and operator of the restaurant known as “bartaco” in Port Chester, New York, the retail food establishment that manufactured, distributed, prepared, served and sold the adulterated food that created the risk that injured the Plaintiffs. At all material times, the Defendant was and is the manufacturer, distributor, preparer, server and seller of the adulterated food product, which food product reached its intended consumers without substantial change from the condition in which it was sold by the Defendant.

42. The Defendant is subject to liability to the Plaintiffs for its breach of express and implied warranties made to the Plaintiffs with respect to the food products sold to those patrons, including the implied warranty of merchantability. Specifically, the Defendant expressly warranted, through its distribution and sale of food to the public, and by the statements and conduct of its employees and agents, that the food it manufactured, distributed, prepared, served and sold to its patrons was fit for human consumption, and not otherwise adulterated or injurious to health.

43. The Plaintiffs allege that the Defendant breached the warranty of merchantability because:

- a. Food sold by the Defendant and consumed by the Plaintiffs, which was adulterated with HAV and related filth and adulteration, would not pass without exception in the trade, and
- b. The adulterated food manufactured, distributed, prepared, served and sold by the Defendant and consumed by the Plaintiff was not fit for the uses and purposes intended by either the patrons or the Defendant, *i.e.*, human consumption.

44. The Defendant owed a duty to the Plaintiffs to manufacture, distribute, prepare, serve and sell food that was not adulterated, was fit for human consumption, was reasonably safe in construction, and was free of pathogenic viruses or other substances injurious to human health. The Defendant breached this duty.

45. The Defendant owed a duty to the Plaintiffs to manufacture, distribute, prepare, serve and sell food that was fit for human consumption, and that was safe to the extent contemplated by a reasonable and ordinary consumer. The Defendant breached this duty.

46. The Plaintiffs became ill and were diagnosed HAV-positive after exposure to food, manufactured, distributed and sold by the Defendant, and are thus persons who the Defendant might reasonably have expected to use, consume or be affected by its adulterated food products.

47. Because the food that the Plaintiffs purchased and consumed was adulterated, not fit for human consumption, not reasonably safe in design and construction, lacked adequate warnings and instructions, and was unsafe to an extent beyond that contemplated by the ordinary consumer, the Defendant breached both express and implied warranties, and is liable to the

Plaintiffs for the harm proximately caused to them by its manufacture, distribution and sale of adulterated and adulterated food products.

V.

SECOND CAUSE OF ACTION
(Negligence)

48. The Plaintiffs repeat and reallege all prior paragraphs as if fully set forth herein.
49. The Defendant manufactured, distributed, prepared, served and sold a food product that was adulterated, not fit for human consumption, and that was not reasonably safe as designed, manufactured, or sold.
50. The Defendant was negligent in the manufacture, distribution, preparation, service and sale of a food product that was adulterated with HAV, not fit for human consumption, and not reasonably safe because adequate warnings or instructions were not provided.
51. The Defendant had a duty to properly supervise, train, and monitor its employees, or the employees of its agents or subcontractors, engaged in the preparation of its food products, to ensure compliance with the Defendant's operating standards and to ensure compliance with all applicable health regulations. The Defendant failed to properly supervise, train, and monitor its employees engaged in the manufacture, distribution, preparation, service, sale and delivery of the food product the Defendant sold to its patrons, and thus breached that duty.
52. The Defendant owed a duty to the Plaintiffs to exercise reasonable care in the manufacture, distribution, preparation, service and sale of its food products, as it was reasonably foreseeable that the Defendant's manufacture and sale of food products adulterated with HAV would cause injury and harm to all persons exposed to HAV. The Defendant has breached that duty, and thereby caused injury to the Plaintiffs.

53. The Defendant was negligent in failing to require its employees to obtain HAV immunizations, and in allowing one or more employees to work while infected with HAV in violation of 10 NYCRR § 14-1.10.

54. The fact that the Plaintiffs became infected as a result of the consumption of food and drink at the Defendant's restaurant conclusively demonstrates the failure of the Defendant to prevent the transmission of HAV by way of adulterated food, drink, and surfaces.

55. The Defendant's negligent acts and omissions have caused the Plaintiffs physical injury, emotional distress, pain and suffering, reasonable fear of injuries and harm, and related general and special damages.

VI.

THIRD CAUSE OF ACTION (Strict Liability)

56. The Plaintiffs repeat and reallege all prior paragraphs as if fully set forth herein.

57. The Defendant manufactured, distributed, prepared, served and sold a food product including HAV-adulterated products, that caused Plaintiffs' confirmed HAV illness. Thus, the Defendant is strictly liable for the Plaintiffs' injuries and resulting damages .

58. The products that the Defendant manufactured, distributed, prepared, served and sold were, at the time of distribution, manufacture or sale, in a condition that a consumer would not reasonably contemplate, including being HAV-adulterated, and were in a condition that rendered the products unreasonably dangerous for their ordinary and expected use.

59. The products that the Defendant manufactured, distributed, prepared, served and sold, and that the Plaintiffs purchased and consumed, as described previously, were expected to reach consumers, and be consumed by them, without substantial change. The Plaintiffs used the product in the manner expected and intended, including consumption.

60. The Plaintiffs suffered the injuries alleged above as a direct and proximate result of their contact or consumption of adulterated, defective food products that the Defendant manufactured, distributed, prepared, served and sold.

VII.

DAMAGES

61. The Plaintiffs have suffered general and special, incidental and consequential damages as the direct and proximate result of the acts and omissions of the Defendant, which damages shall be fully proven at the time of trial. These damages include, but are not limited to: damages for lost wages; medical and medical related expenses; travel and travel-related expenses; pain and suffering; emotional distress, fear of harm and humiliation; physical pain; physical injury; and all other ordinary, incidental and consequential damages as would be anticipated to arise under the circumstances.

VIII.

PRAYER FOR RELIEF

WHEREFORE, the Plaintiffs prays for the following relief:

- 1) That the court award the Plaintiffs judgment against the Defendant for such sums as shall be determined to fully and fairly compensate them for all pain and suffering, general, special, incidental and consequential damages, including but not limited to lost wages and medical expenses respectively incurred by them as the direct and proximate result of the acts and omissions of the Defendant;
- 2) That the court award the Plaintiffs their respective costs, disbursements and reasonable attorneys' fees incurred;

- 3) That the court award the Plaintiffs the opportunity to amend or modify the provisions of this complaint as necessary or appropriate after additional or further discovery is completed in this matter, and after all appropriate parties have been served; and
- 4) That the court award such other and further relief as it deems necessary, just and proper.

Dated: December 7, 2017

UNDERBERG & KESSLER LLP

By: _____

Paul V. Nunes, Esq.
Underberg & Kessler LLP
300 Bausch & Lomb Place
Rochester, New York 14604
Telephone: (585) 258-2800
Fax: (585) 258-2821
Email: pnunes@underbergkessler.com

Of Counsel: (pending admission pro hac vice)

William D. Marler, Esq.
MARLER CLARK, L.L.P., P.S.
1301 Second Avenue, Suite 2800
Seattle, Washington 98101
Telephone: (206) 346-1888
Fax: (206) 346-1898
Email: bmarler@marlerclark.com

Attorneys for Plaintiffs