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DECLARATION OF PATRICK MURPHY, MD, Ph.D.

1. My name is Patrick Murphy, MD, Ph.D., and I reside in Baltimore, Maryland.
2. I am a full professor of medicine at Johns Hopkins University, specializing in Infectious Diseases. My training and experience includes use of chloramphenicol to treat infectious diseases and the associated risks and benefits of this drug product.
3. I have had the opportunity recently to conduct an independent review of the literature on chloramphenicol toxicity, the underlying chemistry, what is known about the relationship between chloramphenicol and the development of aplastic anemia, and the risk of development of chloramphenicol-related aplastic anemia as related to the general risk of aplastic anemia from other causes or from unknown causes.
4. I have also been informed that the U.S. Food and Drug Administration has recently instituted testing of imported crabmeat using liquid chromatography electrospray mass spectroscopy (LCEMS) that purports to detect chloramphenicol residues and has instituted a policy under which detection of residues at the 0.5 ppb results in detention of imported crabmeat.
5. I am also aware that chloramphenicol is a naturally-occurring biological compound produced by soil organisms, *Streptomyces spp.*, and thus is likely to be found at very low levels in coastal waters subject to run-off from nearby soil. It is therefore not possible to eliminate natural exposure to extremely low levels of chloramphenicol.
6. I am also aware that the LCEMS method cannot, obviously, differentiate natural chloramphenicol from added or pharmaceutical-type chloramphenicol.
7. I also understand that crabmeat purveyors have a longstanding limit for chloramphenicol content using an inexpensive ELISA test method that precludes importation of

product with more than 5 ppb chloramphenicol and that this limit itself is occasionally surpassed but mostly because of cross-reaction of the test method with chloride-containing sanitizers that are permitted in food stuffs.

8. There is a background rate of cases of aplastic anemia in persons without any known exposure to bone marrow toxins. This baseline incidence of aplastic anemia is approximately 3-6 patients per 1 million population per year. The risk of death from the baseline incidence of aplastic anemia is estimated at 1/500,000 population per year. Most of these cases are of unknown etiology.

9. When a cause is determined, it is most likely to be associated with a viral infection. The most common viral infections associated with aplastic anemia are parvovirus and hepatitis virus; some cases are caused by Epstein-Barr virus and cytomegalovirus. These latter viruses are present throughout the US population on an endemic basis.

10. Aplastic anemia is also associated with chemical exposure. There are two types of association. The first results from exposure to a sufficient dose of the chemical in question. For these chemicals, it is easy to get a good understanding of the relationship of dose to incidence of aplastic anemia and, in most, there is a readily understandable mechanism involving the destruction of bone marrow cells. These chemicals might be called pure bone marrow toxins and would include, for instance, high doses of radiation. For the second type, there is merely an increased incidence of aplastic anemia upon exposure to the chemical – some persons appear not to be susceptible. Thus, for this type, most people can be given the chemical repeatedly even at high doses without developing any damage to the bone marrow. For susceptible people, however, there is a relationship between dosage of exposure and risk of developing aplastic anemia. Thus, even for this second type, there is a de minimis exposure that does not result in any known harm to anyone. Chloramphenicol fits into this category.

11. In identifying the cause of a disease, it is important to avoid confounding by indication. This occurs when there is also an association between two possible causes of a disease. Thus, both viruses and chloramphenicol may cause aplastic anemia in susceptible patients. However, many patients with viral illnesses may be given chloramphenicol. When a person is given a drug, and subsequently is found to have aplastic anemia, the drug is often blamed without further thought. But since there is a background incidence of aplastic anemia, before the drug can fairly be blamed it is necessary to show that people treated with the drug develop aplastic anemia more often than those that are not treated but might have the same incidence of other things, like viral illnesses.

12. The studies that associated the use of chloramphenicol with aplastic anemia were undertaken precisely because there was a perception that patients on therapeutic doses of chloramphenicol developed aplastic anemia more commonly. This perception was tested by comparing the incidence of aplastic anemia in patients on therapeutic doses—a cut-off of 3 grams of exposure was selected—with the general incidence of aplastic anemia. These studies demonstrated that, for chloramphenicol used in doses measured in grams, the incidence of aplastic anemia is about 13 times the background rate. In other words, somewhere between one in 20,000 and one in 60,000 people treated with therapeutic (more than one gram) doses of chloramphenicol will develop aplastic anemia.

13. There have also been studies that have looked at the association of chloramphenicol in the form of eye drops to treat conjunctival infections and aplastic anemia. Eye drops pass into the nasopharynx via the naso-lacrimal duct, and are swallowed. Chloramphenicol eye drops are equivalent to small quantities of chloramphenicol given by mouth.

14. The world's medical literature describes small numbers of cases of aplastic anemia following the use of chloramphenicol eye drops. However, there is no scientific evidence that these

cases of aplastic anemia following chloramphenicol eye drops are due to the chloramphenicol. They may merely represent the background rate of aplastic anemia. There are two opinions in the medical literature that illustrate this dispute. First, some believe that there is really no increased incidence of aplastic anemia from chloramphenicol eyedrops. Second, others believe that there may be an increase, but it is very small and not of the same order as the incidence after oral doses that are given in gram amounts. Thus, opinion is uniform that the risk associated with chloramphenicol eye drops, if there is any, is nothing like the risk of oral therapy with standard therapeutic doses measured in grams.

15. Chloramphenicol eyedrops and ointments are used very extensively in Europe and in Asia. The British National Formulary recommends chloramphenicol as the best available treatment for “pink eye.” Chloramphenicol accounts for more than half of all the prescriptions for ophthalmic antibiotics in both Great Britain and Eire.

16. Although an editorial has been written suggesting that the use of chloramphenicol eyedrops is harmful and should be abandoned,<sup>1</sup> there is in fact no published evidence that the incidence of aplastic anemia is higher in people who use chloramphenicol eyedrops. Two large recent studies bear on this point. A case control study collected cases of aplastic anemia from various countries in Europe and Asia with a total population of 40 million. The total person-years of study was 185 million. 426 cases of aplastic anemia were found, but there was not a single case associated with chloramphenicol eye drops.<sup>2</sup> On the other hand, seven of 3118 case controls had

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<sup>1</sup> Doona M, Walsh JB. Use of chloramphenicol as topical eye medication: time to cry halt? *British Medical Journal* 310, 1217-8, 1995.

<sup>2</sup> Wilholm BE, Kelly JP, Kaufman D, Issargrisil S, Levy M, Anderson T, Shapiro S. Relation of aplastic anemia to use of chloramphenicol eye drops in two international case control studies. *British Medical Journal* 316, 666, 1998.

used chloramphenicol eye drops (and did not have aplastic anemia). Similarly, a review of a British general practice database covering almost half a million people found three cases of aplastic anemia associated with the use of chloramphenicol eye drops. The crude estimate from those figures would be that the risk of aplastic anemia after chloramphenicol eye drops was about one in 150,000.<sup>3</sup> However, detailed examination of those three cases suggested that none of them was actually a case of aplastic anemia associated with chloramphenicol. One was an epileptic boy who was on two anti-epileptic medications which are both known to cause aplastic anemia. He made a complete recovery when the anti-epileptic medications were withdrawn. The second case was a woman who had cirrhosis of the liver, a condition known to be associated with pancytopenia. Her pancytopenia was stable and caused no symptoms. Both of these cases survived for long periods, whereas aplasia due to chloramphenicol is usually fatal. The third patient was a lady who was given chloramphenicol eyedrops and seven days later had a major bleed. She was found to have aplastic anemia, from which she subsequently died. The period of seven days is probably not enough to develop severe thrombocytopenia, and is certainly not enough to develop anemia. It seems likely that the aplastic anemia was already present when she was given the chloramphenicol eye drops, and indeed the ocular infection may have developed because she had very few white blood cells.

17. If the figure of 1 in 150,000 cases of aplastic anemia after chloramphenicol eyedrops were taken as accurate, the risk of aplastic anemia after chloramphenicol eye drops would be 1/3 to 1/10 that of the risk related to a therapeutic dose in grams of chloramphenicol. Since the risk of aplastic anemia from therapeutic or multi-gram doses is 13 times that of the general risk of aplastic

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<sup>3</sup> Lancaster T, Swart AM, Jick H. Risk of serious haematological toxicity with use of chloramphenicol eye drops in a British general practice database. *British Medical Journal* 316, 667, 1998.

anemia, viewed in the worst light, the risk from the reduced dose associated with chloramphenicol eyedrops approaches that of the general population.

18. Chloramphenicol eyedrops are available as a 0.5% solution which means that there is 0.5 grams of chloramphenicol in 100 ml. In one drop of this solution (50  $\mu$ l), there would be 250  $\mu$ g of chloramphenicol. Eyedrops are generally administered several times per day for a period of 7 to 14 days. Thus, a therapeutic dose of chloramphenicol eyedrops would result in an exposure of about 5,250  $\mu$ g of chloramphenicol or 5.25 mg.

19. Notwithstanding these various opinions and anecdotal experiences, there is no scientifically-valid confirmation that chloramphenicol eye drops have a measurably increased risk of aplastic anemia.

20. Under the weak hypothesis that there is some risk from chloramphenicol eyedrops, however, these data (including the original research on risk related to exposure to therapeutic doses) establish that there is a dose relationship to the risk of aplastic anemia from chloramphenicol as follows:

- a. 3 gms = 13 times increased risk (1/20,000 to 1/60,000)
- b. 3 mgs = 3 times increase risk (1/150,000)

[Baseline or background risk of death from aplastic anemia = 1/500,000]

21. If the research and reports on chloramphenicol eye drops are more carefully reviewed, however, most of the cases can be discarded as unrelated to chloramphenicol exposure. In that event, the following risk relationship would be true:

- c. 3 gms = 13 times increased risk (1/20,000 to 1/60,000)
- d. 3 mgs = same risk (1/500,000) as baseline

[Baseline or background risk of death from aplastic anemia = 1/500,000]

22. While it is often stated that there is no relation between the dose of chloramphenicol and the development of aplastic anemia, this is not an accurate statement when addressing sub-therapeutic doses of chloramphenicol. Generally, studies have only looked at variations of therapeutic oral and intravenous doses and, then, have had to estimate dose exposure based on limited and often inaccurate information. Since each therapeutic dose is likely in the range of 13-fold increased risk, it would require extremely precise knowledge of actual dose exposure to identify a difference between, say 13-fold increased risk and 14- or 12-fold increased risk. It is not surprising that such data failed to yield a dose-response curve. The fact that chloramphenicol eye drops are either safe or minimally dangerous, however, means that there is in fact a dose-response curve for chloramphenicol and its relationship to aplastic anemia. It is my belief that chloramphenicol eye drops are safe based on a careful review of the reports and research and I believe that anyone with similar credentials as mine who carefully reviewed this literature would come to the same conclusion.

23. Thus, there is a low dose of chloramphenicol which is safe and it is likely to be close to, at, or just below the dosage available in chloramphenicol eyedrops (5 mg).

24. Chloramphenicol is also a naturally-occurring substance, and there are likely to be constant but extremely low levels of exposure to chloramphenicol that might be uncovered by sophisticated testing such as that recently instituted by FDA. The amounts of natural chloramphenicol in crabmeat, if any is present, are far below the amounts present in eye drops.

25. I am aware that the new test for chloramphenicol residues in Asian crabmeat may not measure chloramphenicol accurately. I am also aware that chloramphenicol is produced by

*Streptomyces spp* in soil, and that it may be that American crabmeat is similarly contaminated, as shown by positive test results in unspiked samples that were disregarded by FDA.

26. If I were to assume, however, that the test does measure chloramphenicol accurately, and that all Asian crabmeat contains 5 ppb chloramphenicol, a large crabcake made entirely of Asian crab would then contain a maximum dose of 1 µg of chloramphenicol.<sup>4</sup>

27. To reach a dose with *possibly* increased risk (5 mg), an individual would have to consume 5000 such crabcakes. By eating three ½ lb crabcakes per day every day, an individual would reach the threshold of increased risk in 1,667 days, or approximately 4.5 years. If the crabcake is only 50% meat, as is usual, it would take 9 years of this restricted diet.

28. The doses ingested with crabmeat, assuming that the test measures only chloramphenicol, that all crabmeat is maximally contaminated, and that the person was on a diet of only crabmeat, are tenfold less than the dose of chloramphenicol in one eye drop.

29. I do not believe that such doses threaten the health of the American public.

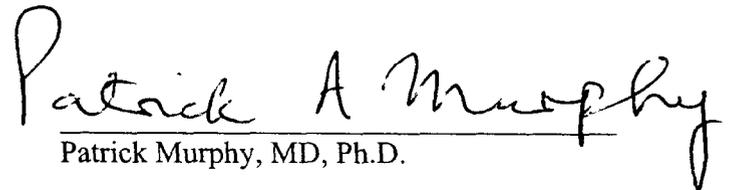
30. I am not aware of any scientific data, after a thorough search, that suggests that exposure to a dose of less than 1 µg of chloramphenicol represents a risk to human health or is likely to increase the incidence of aplastic anemia.

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<sup>4</sup> A 200 gram crabcake (1/2 lb) represents 200,000,000 µg. At 5 ppb, such a crabcake would contain 1 µg of chloramphenicol.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Executed on 5<sup>th</sup> March, 2003.

  
Patrick Murphy, MD, Ph.D.

## CURRICULUM VITAE

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### CURRICULUM VITAE:

FROM 1943 to 1954      St. Edwards Grammar School, Liverpool.  
FROM 1954 to 1956      Liverpool University Medical School  
FROM 1956 to 1957      Liverpool University Department of Physiology.  
FROM 1957 to 1960      Liverpool University Medical School.  
FROM 1960 to 1961      House Physician to Lord Cohen of Birkenhead, Professor  
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FROM 1961 to 1962 The Johns Hopkins Hospital, Assistant Resident,  
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FROM 1962 to 1963 Mossley Hill Hospital, Liverpool, England, Senior  
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FROM 1963 to 1964 Radcliffe Infirmary, Oxford, England, Registrar to  
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**SOCIETIES:** 1960 Liverpool Medical Institution  
1963 Royal College of Physicians (Fellow 1984)  
1976 Infectious Disease Society of America  
1976 American Federation of Clinical Research  
1982 American Association of Immunologists



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