IN THE HIGH COURT OF SOUTH AFRICA
SOUTH GAUTENG HIGH COURT, JOHANNESBURG

Case No:

In the matter between:

BANGUMZI BENNET BALAKAZI	**First Applicant**
WATU LIVINGSTONE DALA	**Second Applicant**
ZWELAKE DALA	**Third Applicant**

and

ANGLOGOLD ASHANTI LIMITED **(Registration number 1944/017354/06)**	Respondent

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FILING SHEET

KINDLY FIND UNDERCOVER hereof the affidavit of Dr. Richard Clapp.

Dated at JOHANNESBURG on the 20TH day of AUGUST 2012.

ABRAHAMS KIEWITZ ATTORNEYS

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TO: **THE REGISTRAR**
South Gauteng High Court
JOHANNESBURG

AND TO: **ANGLOGOLD ASHANTI LIMITED**
Respondent
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AFFIDAVIT OF RICHARD CLAPP, D.Sc.

I. OVERVIEW

1. I am an epidemiologist with over 30 years of scientific experience researching, studying and publishing on causes of human disease, primarily cancer.

2. This Affidavit is submitted at the request of attorney Charles Abrahams of the law firm of Abrahams Kiewitz.

3. I was asked to provide and opinion about the risk of silicosis, pulmonary tuberculosis, silicotuberculosis, chronic obstructive lung disease ("COLD"), and lung cancer in silica-exposed gold miners in South Africa.

4. As I describe in greater detail in this declaration, it is my opinion to a reasonable degree of scientific certainty that:

a. Several lines of evidence in humans and laboratory animals lead to the conclusion that chronic exposure to quartz dust containing respirable silica causes silicosis, silicotuberculosis, COLD, and lung cancer and that this exposure contributes to pulmonary tuberculosis.

b. Airborne average concentration of respirable silica of greater than 0.025 mg/m3 for an eight-hour day is widely recognized as hazardous to workers' lungs.

c. In reaching these opinions and conclusions I have relied on peer-reviewed scientific literature, in addition to my education, experience and training. In doing so, I applied the Bradford Hill guidelines for assessing causation. This methodology is discussed in subsequent paragraphs of this declaration.

II. PROFESSIONAL QUALIFICATIONS

5. I am an epidemiologist specializing in the study of cancer and other diseases

caused by toxic chemicals and other environmental agents. I received the BA degree from Dartmouth College in 1967 with a major in biology and received a MPH degree from the Harvard School of Public Health in 1974 with a concentration in what was then called Health Services. In 1989, I received a Doctor of Science degree from Boston University School of Public Health; this degree was in Epidemiology with a specialization in cancer epidemiology. My dissertation was entitled "Methods of Cancer Surveillance," and was based upon data from the Massachusetts Cancer Registry, where I served as Director from 1980 to 1989. In 1988 and 1989, I was also a Visiting Scholar at the Dana Farber Cancer Institute in Boston, where I attended conferences and analyzed cancer incidence data.

6. My role as Director of the Massachusetts Cancer Registry involved establishing a statewide cancer incidence reporting system in order to track the patterns of cancer in communities and among working populations. As part of this work, I examined data on malignancies in communities and among specific populations of Massachusetts workers and veterans. In particular, I participated in a study of cancer incidence by industry and occupation, which identified an excess of testis cancer in those employed in several occupations. I also participated in numerous conferences and scientific meetings and presented results of a variety of cancer studies during this decade.

7. From 1989 to the present, I have been a consultant providing technical assistance to numerous citizens groups and governmental agencies regarding the health effects of toxic exposures.

8. In 1990, I became Adjunct Assistant Professor in the Department of Environmental Health at the Boston University School of Public Health, lecturing in courses such as Cancer Epidemiology, Survey of Environmental Health, and Cancer Prevention as a Public Health Problem. In 1995, I became an Associate Professor and in 2002 I became a full Professor and continued to teach courses, advise Masters' and doctoral students, participate in research and administrative committees as a member of the Faculty until my retirement in 2010. I currently hold the title of Professor Emeritus at Boston University School of Public Health and

Adjunct Professor at the University of Massachusetts - Lowell. I have lectured in courses at other Massachusetts universities, including Harvard School of Public Health, Massachusetts Institute of Technology, Tufts University, and the University of Massachusetts. I have been a guest lecturer in several courses at the University of Cape Town Medical School over the past two decades.

9. As part of my work as a researcher, educator and consultant, I continually read epidemiologic literature in order to keep abreast of the current knowledge in the field of cancer epidemiology and methods for conducting and assessing the statistical meaning of epidemiologic studies.

10. I am a member of several professional societies, including the Society for Epidemiologic Research, the International Society for Environmental Epidemiology, and the American Public Health Association. I have been an Associate Editor of Environmental Health Perspectives and on the editorial board of New Solutions, a policy journal in environmental and occupational health; I have served as a peer reviewer of articles submitted to several scientific journals, including the New England Journal of Medicine, Cancer Research, Environmental Research, Statistics in Medicine, Environmental Science and Technology, International Journal of Cancer, and Public Health Reports.

11. I have been a member of several scientific advisory panels and I have testified before three committees of the U.S. Congress regarding the health effects of toxic exposures to military veterans. I have made numerous scientific presentations on this topic.

12. I have over 30 years of experience investigating, obtaining, interpreting and evaluating epidemiologic evidence, as a writer, reviewer, researcher and teacher with his special area of emphasis being cancer epidemiology.

13. My *Curriculum Vitae* provides additional information regarding my qualifications. A true and correct copy of my *Curriculum Vitae* is attached as **Exhibit A**.

III. METHODOLOGY UTILIZED IN FORMULATING OPINIONS

A. *Materials and Information Reviewed*

14. I have reviewed the following documents, some of which were provided to me by plaintiff's counsel:

a. Peer-reviewed scientific literature and government reports;

b. Authoritative documents from the International Agency for Research on Cancer and the American Conference of Government Industrial Hygienists.

B. *Summary of Methodology*

15. In forming my scientific opinions in this matter, I applied my education, experience and training in the field of epidemiology and in my specialty, the study of cancer caused by toxic chemicals and other environmental agents.

16. I have used my qualifications in considering the set of causation guidelines known as the Bradford Hill viewpoints. As an aid to the Court in understanding how epidemiologists use the Bradford Hill guidelines, attached hereto as **Exhibit B** is an article I co-authored entitled: "Environment and Health: Vital Intersection or Contested Territory?"

C. *Bradford Hill Guidelines*

17. Physicians and scientists concerned with the causes that contribute to human cancer risk routinely use the Bradford Hill viewpoints or "guidelines" as a set of useful tools for drawing scientific inferences and deductions about causation from all the available relevant principles, data, information, and observations.[1]

18. In performing a Bradford Hill analysis of the evidence establishing a causal relationship between lung disease and the silica exposures at issue, I assess the overall coherence, strength, consistency, and biological plausibility of that evidence.[2]

19. These viewpoints are further explained as follows:

a. Strength of the association: The stronger the association the more likely it is to be causal. The reasoning is that unknown confounders (*i.e.*, unknown factors leading to non-comparability) are unlikely to be powerful enough to explain a strong association. It should be stressed that nothing in the Bradford Hill guidelines requires that associations satisfy any particular test of statistical significance in order to show a strong association; a major reason that statistical significance is *not* a requirement is that often the size of the population available for study is simply not large enough for a positive association to reach statistical significance. In this matter, however, numerous studies consistently show moderate to strong associations that are both statistically significant and biologically plausible.

b. Consistency: Is the association seen in one study also seen in other studies using different designs, different populations, and different investigators? If so, it is less likely that the association is the result of some unseen design flaw or chance occurrence. As the literature discussed below in paragraphs xx through xx shows, in the case of occupational exposures to silica and lung abnormalities, there is broad consistency in the findings.

c. Specificity: If the effect is limited to certain kinds of workers and/or certain diseases (*e.g.*, cancer at a specific site), this may be itself a strong

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[1] The historical context of these viewpoints is of interest: Sir Austin Bradford Hill proposed his viewpoints in the 1960s, well before the International Agency for Research on Cancer ("IARC") had begun promulgating its lists and categories. Moreover, he proposed them in the context of whether cigarette smoke – a complex mixture – causes human lung cancer. Years later the United States Surgeon General relied on Bradford Hill's analytical framework when he issued his findings that cigarette smoking causes human cancer.

[2] As many commentators have explained, coherence means that while a precise knowledge of the mechanism of disease is not required before a causal connection can be made, it is important that the alleged causal connection not conflict with generally known facts about the disease. Thus, in addition to the strength and consistency of the epidemiologic and toxicologic evidence in favor of causation, and the evidence showing there was a biologically significant exposure, the overall proof should correspond with and not contradict what is generally understood about how the disease occurs. Mechanistic, toxicologic and epidemiologic evidence on which I rely is strong, consistent and coherent within the meaning afforded these terms by Sir Austin Bradford Hill and numerous commentators.

argument for causation. Specificity is particularly relevant with "black lung" from coal mining or mesothelioma from asbestos exposure, where diagnosis is virtually pathognomonic confirmation of the prior causal exposure. Similarly, the diagnosis of silicosis in exposed workers is strong and specific evidence of prior silica exposure.

d. Relationship in time: Did the "cause" precede the effect? As applied to the issue of general causation regarding silicosis and silica-related lung cancer, in all of the studies I have reviewed, the exposure being examined occurred before the diagnosis of the disease.

e. Biological gradient (dose-response relationship): If we increase the level of exposure, is there an increase in risk? Bradford Hill noted that when a dose-response relationship is seen, it can be a persuasive argument for causality, but if it is not seen, its absence is *not* a persuasive argument against causality. One reason for this is that the dose-response relationship may be more complex than a simple monotonically increasing function of dose, a particularly relevant consideration when the endpoint in question is human cancer.

f. Coherence of evidence: While a precise knowledge of the mechanism of disease is not required, the alleged causal association must not conflict with generally known facts about the disease; the association should have coherence with those facts. Thus with lung cancer, proposed causal associations with silica are biologically plausible, but the relationship between fibrotic lung disease and cancer development is not precisely understood. That said, and while knowledge from animal and laboratory experiments is relevant and can greatly strengthen the hypothesis of causation and may even determine the actual causative agent, lack of such evidence cannot nullify the epidemiological observations in human

beings.[3]

g. "The Experiment": Bradford Hill suggested that it may be possible to find instances where intervention was followed by a decrease in the disease, providing what he called a kind of "natural experiment." The results of such an experiment might support an inference of causation.

h. Reasoning by analogy: Analogy can be a valuable heuristic device. In the case of lung cancer and the question whether occupational silica exposures are causal factors, one relevant analogy is to Dr. Percival Pott's study of scrotal cancer in chimney sweeps. By looking at the occupational environment of young boys with scrotal cancer, Pott identified exposure to what are now known to be carcinogenic polycyclic aromatic hydrocarbons but which in Dr. Potts' era were simply called "soot." The epidemiologic association was strong and consistent even before the carcinogenic properties of 'soot" were demonstrated.

20. Except that the claimed cause must precede the claimed effect in time, Bradford Hill made clear that none of the viewpoints is a prerequisite for the conclusion that there is an actual causal association between a given exposure and subsequent outcome. Moreover, none of these viewpoints can bring indisputable evidence for or against a cause-and-effect hypothesis and equally none can be required as a *sine qua non*, except that the cause must precede the effect. What the viewpoints can do, with greater or less force, is to help answer the following fundamental question: Is there any other way of explaining the set of facts? Is there any other answer equally, or more likely than cause and effect? Finally, the Bradford Hill guidelines are not meant to replace common sense and judgment but to aid them.

21. In my years of work as an epidemiologist I can aver that these guidelines have

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[3] While the scientific community does not and should not expect an agent causing cancer at one site in an animal will necessarily cause it at the same site in humans, if such correspondence is shown, the probative value of the animal data in demonstrating site-specific human carcinogenicity is that much more powerful. The IARC's upgrade of ethylene oxide from a Group 2A to a Group I carcinogen was based upon what IARC refers to as "supporting evidence from other data relevant to the evaluation of carcinogenicity and its mechanisms." IARC includes evidence of genotoxicity (*i.e.*, structural changes at the level of the gene) within the data it considers. In fact, IARC specifically considered evidence of genotoxicity in its decision.

served the scientific community well. Additionally, they are a generally accepted method of determining which particular factors are substantial contributing causes of cancer.

D. *Design of Epidemiological Studies*

22. While many of the issues and distinctions in study design and methodology will seem arcane to non-epidemiologists there are some concepts that are beyond dispute.

23. To assess potential causal associations between a chemical or physical agent and a cancer or disease, one should review and be familiar with the pertinent literature. If epidemiologic data are available that are germane to the topic, such a review should customarily include evaluation of individual epidemiologic studies for considerations including study design, methodology, and analysis of results. If an epidemiologic study reports an association between potential risk factor "A" and a given cancer but does not collect, analyze or control for potential risk factor "B" for that cancer then that study provides no evidence at all from which to rule out factor "B" as a potential risk factor or contributing cause.

IV. AN EPIDEMIC OF OCCUPATIONAL LUNG DISEASE IN SOUTH AFRICAN GOLD MINERS

24. Multiple investigators have characterized the toll of lung disease in South African gold miners as an "epidemic," going back to the start of the 20th Century and continuing to the present.

25. Dr. G.W.H. Schepers, a South African doctor who monitored the occurrence of disease at the South African Silicosis Medical Bureau from 1944-1956, gave a paper in 1963 and explained that "almost 50 percent of the black miners would die of tuberculosis," and many would suffer both "tuberculosis combined with silicosis." (McCulloch, 2009).

26. Doctors Murray, Davies, and Rees of the South African National Institute of Occupational Health (NIOH) and the University of the Witwatersrand have recently stated that, "South African miners face an epidemic of occupational lung disease," and summarized 10

studies of different design evaluating health outcomes in white and black miners, as well as living and deceased miners, all showing significant proportions of the populations studied suffering from silicosis. (Murray, 2011).

27. Dr. Churchyard and a group from the School of Public Health and Family Medicine, University of Cape Town, studying silica exposure and black gold miners found the data "reaffirm the existence of a significant epidemic of silicosis in the industry." (Churchyard, 2004).

28. Dr. Davies summarized the topic of silicosis and tuberculosis among gold miners by calling the diseases "epidemics" amounting to "a public health disaster of unprecedented proportions." (Davies, 2006).

29. Silica exposure has been shown to cause several occupational lung diseases in South African gold miners, including silicosis, pulmonary tuberculosis, silicotuberculosis, COLD, and lung cancer.

A. *Silicosis*

30. The recognition of silica dust as a cause of lung disease extends back into antiquity (Harley and Vallyathan, 1996). Some of the earliest cases were apparently described by Agricola, in the first century AD, and Paracelsus in the 1500s (see Taylor, 1938). The occupational origins of silicosis were noted two centuries later by Ramazzini, considered by many to be the founder of occupational medicine. It was further described as a pneumoconiosis or "dust disease" of the lung in 1866 by von Zenker (Zenker, 1866) and Visconti (Visconti, 1870). Studies published in the early part of the twentieth century documented increased silicosis and tuberculosis in Australian gold miners (Nelson, 1929) and South African miners (Miners Phthisis Bureau, 1933, cited in Taylor, 1938).

31. An animal model for the production of silicosis from inhalation of free silica in air was published in 1934 (Gardner, 1934), and clinical observations and lung x-rays showed typical fibrotic patterns in South African miners at approximately the same time (Simson, 1930). Clinical symptoms and criteria for differential diagnosis of silicosis were summarized by Taylor

in the same decade (Taylor, 1938). The classical picture of silicosis was summarized recently by Greaves as comprising central nodules surrounded by fibrosis and inflammatory cells in both the lungs and nearby lymph nodes (Greaves, 2000). In advanced cases, the lung tissue may be so damaged that it results in respiratory failure, heart failure and death. Silicosis is a latent disease. There is no cure for the disease and it can be fatal.

32. Silicosis is a progressive disease. Extremely high exposure to fine silica particles can lead to rapid onset of massive fibrosis and death. This condition is referred to as acute silicosis, although the more common scenario is silicosis due to chronic and lower-level exposure. One of the largest industrial disasters in the United States, in which acute silicosis killed hundreds who worked on the Hawk's Nest Tunnel in West Virginia, began in the late 1920s and began to be documented in the 1930s (Cherniack, 1986). The incidence of silicosis in miners is related to the dose and concentration of silica breathed by the miner, and thus is related to the level of dust control and working conditions in the mines.(McCulloch, 2009 p.838; Burke, 1977 p.148-157; Churchyard, 2004 p. 811).

33. A recent summary of the world literature (ACGIH, 2010) concluded that current exposure limits for crystalline silica of 0.05 mg/m3 of respirable dust are insufficiently protective of workers' health. The health risks cited in this authoritative review included silicosis, silicotuberculosis and lung cancer, and the report recommended that industrial hygienists seek to control worker exposure to the level of 0.025 mg/m3.

34. Studies documenting excess rates of lung disease in South African miners date back to the start of the 20th Century. Burke and Richardson documented "six major investigations into the health conditions of miners on the Rand and in Cornwall." (Burke, 1977, p. 159).

35. The Chamber of Miners in South Africa in 1902 reported that mortality among miners who had worked in the Rand caused "....considerable alarm..." (Burke, 1977, p. 164).

36. The 1994 Leon Commission Report indicated silica dust conditions present in the mines were probably unchanged from the 1940's up until the Report. (McCulloch, 2009)

37. Dr. Trapido's research indicated as many as 196,000 miners in South Africa and 84,000 more migrant miners from neighboring states have suffered silicosis. (McCulloch, 2009). Others estimate that some 500,000 cases of occupational lung disease meeting the criteria for compensation have occurred, although investigators conclude that the rates of occupational lung disease in this population have not been adequately studied and have been underestimated. (Marks, 2001p.3; Murray, 2011 p.S 68; Rees, 2006 p.59; Ehrlich 2007 p. 11,13;Trapido 1998 p. 305).

38. The U. S. Public Health Service conducted surveys of granite sheds in the late 1920s and 1930s (Bloomfield, 1934) and documented the silicosis prevalence in a group of granite workers. These surveys provided historical exposure information for later studies of mortality in Vermont granite workers (Davis, et al., 1983) that documented dose-response trends of increasing deaths due to silicosis in 969 workers who died between 1952 and 1978.

39. Hnizdo and Sluis-Cremer (Hnizdo, 1993) studied silicosis in 2,235 white South African gold miners who worked between 1940 and the 1970s and found a strong dose-response trend with increasing incidence of silicosis in those followed to 1991. The highest cumulative silica exposure was associated with a 77% risk of silicosis in these workers.

40. Steenland and Brown (Steenland and Brown, 1995a) sought to further quantify the silica and silicosis exposure-response relationship in a cohort of 3,330 underground miners in a South Dakota gold mine. Their results were consistent with the South African findings and showed a 68 to 84% risk of silicosis in the highest exposed miners. These authors further concluded that the occupational silica exposure standard of 0.09 mg per cubic meter would result in lifetime risks of death that were unacceptably high.

41. A similar analysis of the exposure-response relationship between crystalline silica and silicosis risk in the diatomaceous earth industry (Park, et al., 2002) came to a similar conclusion. The lifetime risk of death due to silicosis under current occupational exposure limits was unacceptably high, based on the findings in workers in this industry.

42. A pooled analysis of data from workers in six cohorts ('t Mannetje, et al., 2002)

examined deaths due to silicosis in various industries in three countries. This pooled analysis did not include South African gold miners because of the way silicosis was recorded in South Africa differed from the conventions used in other countries. The results indicated that cumulative risk of death due to silicosis from 0.10 mg per cubic meter silica exposure over a working lifetime was 13 per 1,000 workers. The prevalence of silicosis in black South African goldminers was estimated by Churchyard and colleagues (Churchyard, et al., 2004) in a cross-sectional survey of 520 migrant contract workers. The average respirable dust exposure concentration was 0.37 mg per cubic meter and the prevalence of silicosis was approximately 19% in this study. The authors note that their results indicate that the international exposure limit of 0.1 mg per cubic meter is not protective against silicosis. Recent 2007 autopsy studies indicate silicosis rates of 32% in black gold miners. (Murray, 2011 p. S 67).

43. In sum, the epidemiological evidence shows that silica exposure in South African gold miners has caused epidemic levels of silicosis, leading to deaths and disease over a period of time extending back to the turn of the 20th Century and continuing to the present. A recent overview describes the current extent of gold miners phthisis as "a severe epidemic." (Ehrlich, 2007)

B. *PULMONARY TUBERCULOSIS AND SILICOTUBERCULOSIS*

44. Numerous investigations have indentified that exposure to silica dust and a silicosis diagnosis are strong risk factors for pulmonary tuberculosis. (Hnizdo, 1998).

45. As early as 1909, deaths from "phthisis" (defined as wasting away, especially the upper body, presumably from tuberculosis) accounted for 9.6% of all deaths in the Transvaal (1,228 deaths), and the rates were particularly high in "coloured males." (Burke, 1977)

46. Pulmonary tuberculosis is recognized as an occupational lung disease in miners exposed to silica. (Hnizdo. 1998) Pulmonary tuberculosis is a latent, bacterial disease which causes infection in the lungs . The disease can be treated if detected early on, although if not properly treated it can be fatal. (Burke, 1977 p. 148; Hnizdo, 1998 p. 500-502).

Silicotuberculosis is a disease including a diagnosis of both silicosis and tuberculosis . (Greaves, 2000 at 246).47. Hnizdo showed that a silicosis diagnosis increases the risk of pulmonary tuberculosis by four times after adjustment for cumulative dust and tobacco. (Hnizdo, 1998)

48. Silica dust also increases the risk of pulmonary tuberculosis in the absence of a silicosis diagnosis, and it continues to elevate the risk after exposure ceases. (Hnizdo, 1998; Murray, 2011, p. S 67)

49. The risks of pulmonary tuberculosis, silicosis, and more recently HIV infection, combine multiplicatively and have acted to cause the "highest recorded rates of TB" in the world to exist among South African gold miners. (Murray, 2011, p. S 67)

50. South African commissions of inquiry in 1902 showed that the rate of mortality from lung disease in South African gold miners far exceeded the death rates in England, India, America and Brazil; and that the average age of death among miners in the Transvaal was 36.3 years old. (Davies, 2006)

51. William Crawford Gorgas, the Surgeon General of the U.S., was invited by the Chamber of Miners to inspect the mines at Witwatersrand in 1914, and found the tuberculosis rates among miners to be approximately four times larger than in the workforce involved in building the Panama Canal. (Davies, 2006)

52. The Medical Committee on Tuberculosis, formed by the Chamber of Mines in 1925, studied the migrant labor systems and found that 60% of the repatriated miners ("repatriated boys") had died within two years of repatriation. (Davies, 2006)

53. This pattern of tuberculosis and death among South African miners continued into the 1960's. Chamber of Miners records published in 1967 showed rates of tuberculosis double that of the most severely stricken countries in the world. (Davies, 2006)

54. The epidemiological evidence shows that silica exposure in South African gold miners has caused epidemic levels of pulmonary tuberculosis and silicotuberculosis, leading to death and disease over a period of time extending back to the turn of the 20th Century and

continuing to present.

C. CHRONIC OBSTRUCTIVE LUNG DISEASE

55. Chronic obstructive lung disease ("COLD") has been associated with exposure to silica in South African gold miners. (Hnizdo ,1993 p. 448; Trapido, 1998 p.301; Girdler-Brown, 2008 p.640, 644, 646; Roberts 2009 p.38).

56. COLD is a condition characterized by reduced lung function following chronic inflammation or damage to lung tissue. It is a latent disease that can be treated to prevent further loss of lung function. (Hnizdo, 2003).

D. LUNG CANCER

57. The evidence regarding the ability of respirable silica to cause lung cancer is also voluminous. Lung cancer is a latent disease and can be fatal. Hnizdo and Sluis-Cremer (Hnizdo, 1991) examined lung cancer risk in 2,209 white South African gold miners who started work in the period 1936-1943 and were followed until the end of 1986. These authors were able to adjust for the effects of tobacco smoking and found that those long-term miners in the highest category of cumulative exposure to silica dust had a three-fold higher risk of death due to lung cancer than those in the lowest category.

58. Lung cancer risk in silica-exposed workers in China was investigated in a nested case-control study (McLaughlin, et al., 1992). This study examined exposures in 316 male lung cancer cases and 1,352 controls in various industries in South Central China. The highest lung cancer risk was in workers in tin mines, which increased in a dose-dependent fashion, and iron-copper, but no excess risk was found in workers tungsten mines or the pottery industry. These authors concluded that their findings provide "some evidence for a silica lung cancer association" but that was hard to disentangle this from the effects of other exposures. DeKlerk and colleagues published the results of a preliminary study of lung cancer mortality on Western Australian gold miners (DeKlerk, et al., 1995). Although based on relatively few lung cancer deaths, the study reported a greater than two-fold increased risk of dying of this cause in those

who worked more than forty years in underground employment. The authors conclude that further follow-up will provide more definitive results.

59. A subsequent evaluation of the carcinogenicity of crystalline silica was carried out by the International Agency for Research on Cancer (IARC, 1997). This monograph evaluated the evidence from animal studies, studies of humans in various industries, and mechanistic data about the carcinogenic effect. The summary evaluation was that there was "sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources."

60. Data on lung cancer in South African, Australian and U.S. gold miners (Hnizdo, 1997; DeKlerk, 1998; Steenland, 1995) and other silica-exposed workers in Finland (Koskela, et al., 1993), the U.S. and China were combined in a pooled exposure-response analysis by Steenland and colleagues (Steenland, et al., 2001). The pooled results included over 1,000 lung cancer deaths and showed a positive exposure-response relationship between silica exposure and lung cancer risk that added to the weight of evidence previously reviewed by IARC.

61. In sum, the epidemiological evidence shows that silica exposure in South African gold miners has caused lung cancer, leading to death and disease over a period of time extending back well into the 20th Century and continuing to present.

CONCLUSION

62. Based on the evidence set forth here, it is clear that there is strong, coherent, consistent and biologically plausible evidence that occupational exposure to silica in South African gold mines causes silicosis, silicotuberculosis, chronic obstructive lung disease, and lung cancer; this exposure also contributes to pulmonary tuberculosis in gold miners. My opinions are based upon all of the foregoing and my knowledge of all of the relevant scientific disciplines.

Richard Clapp

Richard Clapp D.Sc. MPH

I certify that:

The deponent acknowledged to me that he knows and understands the contents of this Affidavit; he has no objection to taking the prescribed oath; and he considers the prescribed oath to be binding on his conscience. The deponent thereafter uttered the words: "I swear that the contents of this declaration are true, so help me God." The deponent signed this Affidavit in my presence at the address and date set out hereunder.

Signed on this 9th day of July, 2012 at Boston, Massachusetts:

STEPHEN E. FOWLER
Notary Public
COMMONWEALTH OF MASSACHUSETTS
My Commission Expires
April 1, 2016

Notary Public

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