UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

BEFORE THE ATOMIC SAFETY AND LICENSING BOARD

In the Matter of	)	
	)	
CAROLINA POWER & LIGHT COMPANY	)	
AND NORTH CAROLINA EASTERN	)	
MUNICIPAL POWER AGENCY	)	Docket Nos. 50-400 OL
	)	50-401 OL
(Shearon Harris Nuclear Power	)	
Plant, Units 1 and 2)	)	

JOINT INTERVENORS RESPONSE TO APPLICANTS' INTERROGATORIES ON JOINT CONTENTION II (First Set)

This response is being filed pursuant to an extension of time worked out with Applicants' attorneys.

Response To General Interrogatories

1(a) The only persons who have first-hand knowledge of these facts are the authors of the reports cited in Joint Contention II and it's references.

(b) Those facts are set forth in the contention, and it's references, including contentions superseded by Joint Contention II.

(c) The allegations, facts, and authors of the reports containing those facts are set out together at various places in Joint Contention II

2(a) None other than Joint Intervenors.

3(a) None so far.

(b) N/A

(c) N/A

4(a) Documents are identified in the contention. Other documents are identified in response to the individual interrogatories which follow.

(b) See response to 4(a).

5(a) Such documents will be identified in specific responses below.

(b) The ones they appear in, or are referenced in.

6(a) Joint Intervenors' general knowledge, experience, and information.

(b) All of them.

7(a) None identified so far.

(b) N/A

Responses To Sepcific Interrogatories On
Joint Contention II (Health Effects)

II-I All cancers; leukemia (in so far as it is considered distinct from cancer); non-dominant genetic diseases of all types.

II-2(a) Joint Intervenors have not made this estimate. At a minimum, a correct estimation should be based on (i)radioactive releases from the plant without the assumption that systems will be functioning normally (the limiting "normal operation" radioactive emissions and material releases from Harris should be the maximum ones which do not force the plant to shut down); (ii)dose estimates should fully take account of the variables described in Joint II(e) including the tendency of radionuclides on coal fly ash of small diameter (under 2 microns) to remain in human lungs, greater absorption and adsorption of radionuclides in soils and growing plants (including gardens) near the plant, and the effects of using higher estimates for radionuclide transfer and transport factors (e.g.

those reported in NRC translation 520 and it's references, but in any case the higher or highest ones reported for each such nuclide); (iii)analysis of the effects of internal emitters which is most conservative (i.e. assumes harm at the lowest radiation levels reported), fully takes into account all decay products of a given radionuclide, and their chemical and radio-toxicity, using the highest estimates of relative biological effectiveness (RBE) of each type of radiation emitted by each such nuclide; (iv)estimates health effects of low-level radiation by a supra-linear model (i.e. one taking account of the higher amount of health effects per rad or per rem at low-levels of radiation, e.g. as demonstrated by the Mancuso-Stewart-Kneale (MSK) and Gofman analyses of Hanford data); (v)takes full account of the variation in ages and other conditions which increase sensitivity to radiation damage and its health effects (e.g. allergies, diabetes, etc. as demonstrated in the work of Bertell and previously referenced to Applicants by W. Eddleman re his contention 37B) by assuming the higher-vulnerability groups in the population, (e.g. fetuses and embryos) actually have the vulnerability to radiation-induced health effects that the work of the above authors and others (whichever are most conservative, i.e. find the highest effect or vulnerability for a given group in the population)indicate; which take the cancer-induction estimates per unit dose of Gofman, Radiation and Human Health, chapters 8, 9 and 10 (especially Figure 5 and Tables 20 through 30), and the supporting evidence for these in that book, or any higher estimates by Mancuso-Stewart-Kneale or others.

(b) Joint Intervenors do not contend the above is a complete analysis. The basis of the above is that when estimating radiation risks, a conservative approach to all parts of the process whereby

nuclides and radiation are made in the nuclear plant, released to the environment, get to and into persons, and have effects on those persons (e.g. cancer and genetic diseases), is that which takes the largest effect, release, transport or other factor or estimate for each step. Since we don't know empirically exactly how radiation causes cancer, or where all the powerplant releases (radiation and radioactive materials) go in the environment, we should make estimates conservatively. Cancers and genetic defects caused by radiation from nuclear plants (including radioactive material released from them) do not come with convenient tags identifying their source.

II-3 Joint Intervenors are aware that a number of authorities are questioning the use of a "latency period" for cancers induced by radiation, on the basis that the greater the number of exposed persons studied, the sooner the first cases show up. Joint Intervenors have used the term "latency period" to mean that period after exposure to a cancer-causing agent (such as nuclear radiation) during which one sees, or expects to see, no effects (or no statistically significant effects) on the exposed group being observed.

II-4 In two ways: (1)in the studies of radiation-induced mortality on which the BEIR-III report relies, by not following up for a sufficiently long period to see how many diseases (cancers, genetic defects, etc.) actually occur; (2)as shown in note j on page 199, by assuming a 10-year latent period for cancers in its estimates of numbers of deaths and cancers to be expected in the population. This period should be shorter, for the effects in a large population (see response to #3 above: the latency period idea may not apply). Rossi, in criticizing the report (at pages

278-279 thereof) points out the increased breast cancer mortalities within 5 to 9 years of irradiation, and notes that if any upper limit on the latent period exists, it is over 30 years. While BEIR-III does not use a 30-year limit on cancer induction by radiation, it does not have a 30-year or more follow-up in most of the data it uses on cancers and other diseases/disorders.

II-5 At least part of the observed latency periods stems from the insensitivity of the observation techniques; that is, the cancers have to be of a certain size and/or manifest some effects before they are likely to be discovered and diagnosed. Currently the whole concept of latency periods are coming under question (see response to II-3), thus Joint Intervenors are not certain that there is a "correct understanding" at the moment.

II-6 All forms of cancer have been considered to have a latency period. That is, Joint Intervenors are not aware of any cancer which has appeared immediately upon irradiation. Joint Intervenors do not have a "latency period theory" insofar as we understand that term. As stated above in response to interrogarory II-3, the whole concept of latency period is coming under question.

II-7 One which shows up as a disease if either of a pair of autosomal chromosomes have a defect. Gofman 81, Radiation and Human Health, page 786.

II-8 All genetic defects not dominant, i.e. which can be transmitted without showing up in a person who is a carrier. This includes the irregularly inherited diseases (Gofman 81, pages 790-91, 845-849, and 850-853), the mild mutations referred to by Bertell (The Nuclear Worker and Ionizing Radiation, Am. Industrial Hygiene Assn. Journal 40, pages 395-401 (1979)), chromosomal diseases,

congenital anomalies, anomalies expressed later and constitutional and degenerative diseases (per UNSCEAR, Table 50, page 539, 1977, ref. Gofman page 788.)

II-9 The lowered resistance to diseases, and the higher radiation-induced cancer incidence referred to by Bertell (op cit above) are an appropriate methodology. Failure to achieve full potential (another such somatic effect) is much harder to estimate and Joint Intervenors have not formulated a methodology for dealing with this.

II-10 The "recessive" effects (as defined above) take a long time to show up. Their damage goes out to the end of the human race, as K.Z. Morgan has pointed out, exposing more people to lower levels of radiation actually increases the genetic burden on the population. Joint Intervenors therefore propose that the effect be estimated over a long period, such as the lifetime of nuclear wastes' toxicity above the radiotoxicity of their parent ore (if it had been left undisturbed instead of mined and used in the nuclear fuel cycle). This is about 11 million years (ref Pigford, Nuclear Safety, 1-82, quoting B.L. Cohen).

II-11 First, as the report notes (pp 304) certain human genotypes are at increased risk. This is not included in the report, though increased susceptibility to disease (and cancer) as an effect of radiation exposure has been found or suggested by Bertell (op cit), Bross et al (op cit), Gofman, et al. BEIR chair Radford states (p. 248) that this may have to be taken into account someday (increased susceptibility to radiogenic cancers). BEIR estimates (p. 5) only take into account genetic conditions that are seriously handicapping at some time in the life of the person who has them. Thus, any lesser handicap (including mild mental retardation, increased susceptibility

to other diseases, etc.) is excluded. Inclusion of these problems would, of course, increase the estimates of somatic health effects, both cancers and others. Radford notes (p. 240) that "consistent visible chromosomal abnormalities in the early stages of solid tumors have not been found". (cf. Gofman(851-852): either the damage is unseen and having effects, or the radiation gives visible chromosome effects only inconsistently while causing cancer.) Consider those effects, and estimates of somatic effects rise.

II-12 (a)Gofman, pp. 788-853, begins to approach this. (See especially pages 848-849 for the genetic effects). For somatic effects, further estimates based on the work of Bertell, et al would be required. Joint Intervenors have not made those estimates.

(b)See Gofman and Bertell, ops cit. See also Radford, BEIR pp. 248-9, regarding higher chromosomal abnormalities at low rad. doses.

II-13 It would increase the BEIR estimates. See, e.g., Gofman, op cit, pp 840-853 and information referred to therein. At page 114, BEIR-III says "We have rather arbitrarily chosen the range of 5-50% for this value" (i.e. the mutational component of irregularly inherited diseases). Gofman notes there is no evidence for this assumption. BEIR-III gives none. A 2 to 20 fold increase would result from correcting this baseless assumption.

II-14 (a)See Gofman, pp 788-853, for a better estimate than BEIR's. We do not know that "the correct estimate" has yet been made. BEIR (Table 25) omits in-utero effects from its estimates. These should also be included.

(b)See Gofman, pp 788-853; logic.

II-15 A model which shows a response at low levels of radiation which is above the linear response dose line which would be projected from data at higher levels of radiation exposure.

II-16 A threshold model assumes that there is some level of radiation exposure below which effects do not occur; or in the case of genetic diseases, a threshold model could include one with a low "mutational component". See Gofman '81, pp 846-853.

II-17 A linear model is one that assumes the health effects of radiation are proportional to dose at all levels of radiation: Thus, the effects versus dose "curve" is a straight line. If the effect of low doses is zero or less than the amount projected by the linear model, a model incorporating such a dose-response curve is a "linear-or-less model". If the assumption is zero effect below a certain dose, that is a special case called the threshold model, BEIR-III (p.4) uses a threshold model for fetal malformations caused by radiation.

II-18 Because the effects are supralinear. See MSK studies, Bross & Driscoll (Yale J. Bio. Med. 54:317-328 (1981)), HH Rossi, ibid pp 340-341; Potten, C.S., Int'l J. Radiation Biology (1981), 40:2, 217-225; ICRP publication 18 (1972) pp 28-29 and 32-33. See also Bross, Ball & Fallen, Am. J. Public Health (1979) 69:130-136. This study found a higher level of both heart disease and non-lymphatic leukemias due to X-ray in the 100mrad - 10 rad range. "The most disturbing feature is that, for each of the three age groups, the dosage response curve tends to flatten out in going down to the lowest dosages." (p. 135, emphasis in original). From the actual curves, we can see that the flattening out referred to is that the effect is supralinear, as defined above. (Curves, Figure 3, p. 135)

Dr. Edward Radford of BEIR (critique, p. 248) says that for high-LET radiation the dose response curve may be curvilinear downward. (Alpha and neutrons from internal emitters are high LET: other radiation may be in a given case). Radford also notes that (p. 238) BEIR's lower radiation effect estimates result from a lesser effect shown at high radiation levels (over 100 rad) in the Hiroshima/ Nagasaki sutdies. K.Z. Morgan has also noted some evidence for a supralinear response to low radiation doses. See also Radford, BEIR p. 232, why low-dose radiation could cause more cancers per unit dose.

II-19 It would increase them. BEIR's models are linear or linear plus quadratic models, which thus give the same or higher effect per rem (or rad) of radiation dose at higher levels of radiation exposure. Joint Intervenors have not determined the increase, but a factor of 2 for BEIR underestimates and a further factor of 2 or 3 for the errors in dose in the Hiroshima and Nagasaki populations BEIR-III uses as basis, would be a minimum estimate of the cancer underestimate. The genetic underestimate, as shown by Gofman (op cit above) is likely to be even higher. We can place no limit on the amount of the BEIR-III underestimate at this time.

II-20 Greater specific irradiation, spaced radiation exposure, long-term radiation exposure, higher RBE, exposure of cells damaged by previous irradiation (membranes, chromosomes, enzymes, other parts, DNA), higher LET for alpha and neutron emitters, reducing the effectiveness of the immune system and the body's defenses against disease, killing cells, mutating cells, mutating bacteria or viruses (possibly into more dangerous forms, or ones the body's defenses are less able to handle), greater chemical and radiochemical toxicity, chemical carcinogenesis as well as radiation-induced carcinogenesis

(relying on the idea endorsed by Radford (BEIR-III p. 233) that the "promoting step of radiation carcinogenesis in man (sic) is independent of the initiating event.")

II-21 By NRC, ICRP, the West German government, and others. See, e.g., NRC trans 520, especially at pp 37-90, 90-97, 98-106, and 107-115, 122-128 and references cited therein.

II-22 In some cases, because they haven't been estimated, or haven't been included in BEIR-III and other estimates. For example, BEIR-III at page 469 refers to membrane damage from radiation,

> "The inverse relationship between dose rate and the induction of damage in model membrane systems and the possible relationship of such alterations in biomembranes to carcinogenesis suggests that this phenomemon may be involved in low-does or low-dose-rate effects in living systems. Thus, there is a need for additional studies in this field."

See NRC translation 520 at pages 117-118, detailing these sites of damage and references. The underestimation of RBE and specific radiation dose from alpha and neutron radiation is stated by Morgan (Bull at Sci 9/78), Radford (BEIR-III critique at 247) RBE 10 to 20 for alpha, for lung and liver cancer. The effects that give obvious cancers and immediate handicapping health effects are considered, but other damage isn't well considered. Failure to diagnose cancer (Radford, p. 238-239) also leads to underestimates of the health effects of radiation damage.

These higher RBE's, the difference in radiation actually delivered at Hiroshima and Nagasaki from what the BEIR-III studies were based on (see pp 169-226, especially p. 178), the underestimation

of cancers in that data (Radford, BEIR-III, 238-239), the lack of consideration of the nonlethal effects, or the effects which predispose a person with such damage to other diseases (including cancer), the effects of damage such as increased interstitial and arteriocapillary fibrosis (Casarett, G.W., Similarities Between Radiation and Time Pathology, Adv. Gerontological Research 1:109-163, 1964) ref BIER at p. 507, p. 502, error prone repair mechanisms and weakening of antibody formation (Little, referenced by Radford, BEIR, p. 235), translocations and deletions (etc) so that cells can't form enzymes or can't produce them in needed amounts, the supralinear effect of damage by radiation on DNA (Berek & Hall, referenced by Radford, see pp. 233035 and 235-36), and other radiation effects (Joint Intervenors' research on this matter is not complete) combine to produce greater damage from radiation than has been estimated by BEIR-III, Applicants, or NRC Staff.

II-23 Joint Intervenors have not made such an estimate. The underestimate induced as a result of all the above effects is quite substantial.

II-24(a) DNA controls genetic transmission of information needed for future generations of human beings to develop normally, resist disease, and to become parents of further generations who also need to be healthy, able to develop normally, and able to resist disease. DNA is the basis of the "hardward" necessary for human beings in this and future generations to achieve their fullest potential. Further, DNA information helps keep cells functioning normally in human beings. (All of this is true of DNA of non-human organisms too, with respect to those organisms development, disease-resistance, and achievements and normal function.)

(b) Cell membranes are vital to cell life, to recognition of disease organisms and of other parts of one's own body (e.g. nearby cells), to control of nutrients and waste products in living cells, and to prevention of uncontrolled cell growth (e.g. cancer).

(c) Enzymes are necessary for all forms of human development and virtually all steps in human metabolism. Enzyme defects can cause severe or mild genetic diseases, cancers, and other problems, or death. Thus, if the effects of alpha, beta and neutron radiation (RBE above 1, greater specific irradiation than x-rays, particularly from internal emitters) on the above are underestimated, so are the somatic and genetic health effects (deaths, genetic diseases, cancers, other diseases) resulting from such damage.

II-25 NRC estimates (see reg guides 1.109 and 1.113, and DES's, e.g. for McGuire) appear to only cover the operating life of the plant. Joint Intervenors have not located NRC's public statements, but believe they are similar.

II-26 Any time shorter than the time those radionuclides released from Harris will actually remain radioactive in the environment in any quantity.

II-27 The effects should be estimated for the whole lifetime of the radionuclides released, i.e. as long as the radioactive material remains in the environment -- we'd suggest at minimum 40 half lives for materials released in nanocurie quantifies, 50 half lives for materials released in microcurie quantities, 60 half lives for materials released in millicurie quantities, and 70 half lives for materials released in curie quantities, 80 half lives for kilocurie quantities released, 90 half lives for megacurie quantities

released, and so on. Ideally this estimation should be made until the last nuclide decays into a stable form, and the health effects of that stable form as a chemical should also be included. Any shorter time (e.g. the lifetime of persons near the plant, or the operating life of the Harris plant) is arbitrarily short because the radio-active emissions will still be there in some quantity after that time has expired. To take some obvious examples, I-129 will be around for over a million years; Sr-90 will take about 800 to 1100 years for 30 to 40 halflives; Cs-137 will take rather longer, about 1000 to 1400 years for the same number of half lives. We believe the environmental effects should be estimated over the time that they will be able to occur, i.e. as long as the radioactive materials emitted from the plant remain radioactive.

II-28 The minimal acceptable period is hard to define, but the above defintion (in response to J-II-27) is the one we hold to. We also note B.L. Choen's estimate that the radiotoxicity of material which has been through the nuclear fuel cycle remains more hazardous than its undisturbed parent ore would be, for 11 million years. Thus, no period less than 11 million years would be adequate. The period could be much longer, but we have not made the calculation. Please note that by nuclear fuel "cycle" above we refer to the once-through fuel cycle for Harris, including mining, milling, conversion, enrichment, fabrication, use in a reactor, and waste disposal.

II-29 See response above to II-27 and II-28.

II-30 A greater cancer mortality rate than seen in the absence of the nuclear facility or its releases.

II-31 Millstone, in Connecticut: others, ref. Sternglass: Shippingport, Peach Bottom. We don't have a complete list. Rocky Flats (plutonium, studies of Carl Johnson) should also be included.

II-32 U.S. Public Health Service, Ernest Sternglass, Carl Johnson, and others.

II-33 Those used in the ER and its preparation; those used by NRC in its Regulatory Guides and DESs; See NRC Translation 520 also, especially sections 8 and 9.

II-34 Rainout is the deposition of radionuclides from the air (particles, liquids, solids, gas, aerosol, etc.) through downward movement associated with rainstorms, snow storms, hail, etc. (including downdrafts of wind), whether the materials are dissolved or incorporated into the rain or snow or not.

Hot spots are areas of greater radionuclide deposition, whether caused by rainout, an aerial plume of radioactive material coming to ground in a given place for some time, settling of radioactive particulates or aerosols from an airstream, or by other means.

II-35 Joint Intervenors have not made such a calculation. The local concentration resulting from rainout of radionuclides can be thousands or millions of times greater, at a given spot, than would be expected without rainout.

II-36 These higher concentrations can readily occur in open areas like pastures, yards, playgrounds, vegetable gardens, roads, roofs and parking lots (etc.) resulting in greater internal and external radiation dose to persons in those areas, e.g. through uptake in soil and plants (and animals fed on plants exposed to the radioactivity or grown in exposed soil) through water and runoff, through eating snow or ice (snow cream has been popular in this area

of North Carolina), through inhalation of such particles stirred up in play, by motor vehicles, by later winds, by Brownian motion, by walking or running on such surface, by air handlers and air-moving equipment (e.g. fans, roof air handler intakes, wall air handler and air conditioner intakes), by absorption through cuts or abrasions on the skin, e.g. from falling on such a surface where rainout or a hot spot has increased radionuclide concentration, by transfer of the material to foods in supermarkets, vehicles, gardens or storage in movements of soil, air and water (or any of them), and so on. The greater concentrations thus resulting should be inputs into each further concentration and dosage pathway in the model of radionuclide concentration and resulting dosages of radiation to persons and other creatures in the environment.

II-37 This is adsorption onto the inner and outer surface of coal particulates (which have a very open fine structure in their smaller sizes), either electrostatically or otherwise, or simply being physically inside the coal particulate.

II-38 Basically, electrostatic attachment: through decay of one or more atoms in an aerosol or particulate, or from a radioactive gas decaying into another radionuclide not gaseous itself, or through adsorption of gaseous, aerosol, or particulate radionuclides directly.

II-39 Joint Intervenors do not refer to specific coal plants in Contention Joint-II(e). The smaller coal fly ash particles can travel for extremely long distances, so that we could not rule out coal plants in the Soviet Union or Europe or Japan or China, or any place nearer, as sources of coal particulates in the air near the Harris plant, or in air which contains radioactive materials emitted from the Harris plant.

II-40 Joint Intervenors have not made this calculation; however, the problem is the deposition of these coal particles directly in the deep lung, which is most severe for those below 0.5 micron diameter, but applies to some extent to some particles below 10 microns and many below 2 microns in diameter.

II-41 This concentration occurs not only in humans, but in animals (see, e.g. rabbit lung studies of C. Aranyi et al), and probably occurs in deposition to soils, growing crops and pasture, and directly into open containers and displays of foods both liquids and solid, including plates and drinking vessels. This pathway needs to be fully analyzed for the additional concentrations it produces in persons directly (through their lungs) and indirectly through food animals and other foods. The lung is an important site of chemical synthesis, so that radionuclides from coal particulates could readily be transported throughout the bodies of creatures breathing them. The intake of such particles in liquids, especially by micro-organisms, should also be throughly investigated as a pathway for transport and concentration of radionuclides in the environment and in food chains leading to human beings.

All of this estimation and analysis should be done convervatively, i.e. assuming the higher or highest deposition, retention, transfer and concentration factors, chemical activity and biochemical activity reported in the scientific literature, or in research undertaken to find these effects to the extent they are not currently documented or completely analyzed.

The above response applies to both (a) and (b) of this question, but the analyses should take into account that radionuclides on the outside of coal particulates are more readily removed in the

body or by contact with air or liquids, than those inside the particulates. This difference is not one of adsorption or attachment, but of location.

II-42 This phenomemon is that radionuclides when released from the Harris plant (or any source) into the environment do not completely mix, or uniformly disperse, therein. Thus, for example, if 1 curie of a nuclide is released into a river of 1 billion cc during the release (flow past release point during release) and the theoretical or legal concentration limit is one nanocurie per cc, this release does not satisfy the limit, since mixing and dispersion will be incomplete.

II-43 Basically, reality: patterns and temperature gradients and inversions in air and water, quantum effects, metastability of air and water current patterns, phase change phenomena (including rain, snow, dew, evaporation and condensation, melting, boiling, freezing) irregularities in the environment including the physical surface of the earth, buildings, trees, other plants and growth, the action of chemical and biological agents in air and water, irregularities in air and water currents and flows, including changes in direction and uniformities of direction; the characteristics of the radionuclides themselves, as elements and as physical states (e.g. solid, liquid, gas, aerosol, plasma, charged or uncharged electrically) and their decays into other radioactive elements or into nonradioactive elements; the chemistry of the radionuclides involved; the physical properties of the radionuclides involved; human action and accidents at the plant; timing of releases. weather and water conditions at and after the time of releases; and probably other causes.

II-44 The best way is to track all the Harris emissions (or nuclear plant emissions input to a model) to their ultimate decay into nonradioactive material. This would be difficult, and might require considerable additional research beyond what went into current models.

II-45 Joint Intervenors have not made this calculation.

II-46 Those used in preparing NRC's models (see NRC translation 520). See NRC reg. guide 1.109, 1.113, etc. See also Pisiello study of Kr-85 releases at TMI, and LEAF, Methodologies for the Study of Low-Level Radiation.

II-47 Essentially, all those used by NRC. Examples are in NRC translation 520 pp. 45-55 and otherwise.

II-48 Joint Intervenors have not completed listing these. A partial response is included in the materials cited in II-47, above.

II-49 The impact for each is to lower the concentration of such radionuclide in the environment, including those eventually absorbed by human beings, and thus to underestimate radiation dose to humans and other animals in the environment.

II-50 The "noncritical ones": See LEAF, Methodologies for the Study of Low-Level Radiation.

Request for Production of Documents: Joint Intervenors possess copies of some of the documents referenced above and will make them available to Applicants for inspection and copying at a mutually agreed time and place.

I, M. Travis Payne, have prepared these responses with assistance from the other Joint Intervenors. They are true and correct to the best of my knowledge.

This the 16 day of May, 1983.

So sworn,

M. Travis Payne

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CERTIFICATE OF SERVICE

I hereby certify that a copy of the foregoing document has been served by deposit in the United States Mail, first class prepaid, addressed to the parties listed below.

This the 16th day of May, 1983.

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