A ... position as it existed, as it was then, I am looking now at the history that I wrote for—history of preventive medicine in World War Two. From the objectives of research, I describe wherein as follows: the high incidence of sand fly fever among British troops stationed in Palestine and the Middle East since 1939 and its appearance among American troops in the Middle East on the Persian Gulf commands, you see, I didn't remember that but it had already occurred. As well as in the Asiatic theaters in 1942 led the commission of neurotropic virus diseases to undertake experimental studies on this disease. Of course it not at the initiative of the commission. It was at the initiative of the office of the surgeon general of the U.S. Army. The first group to concern itself with this work consisted of Dr. John R. Paul, who was director of the commission, Major Cornelius Phillip, an entomologist and myself, who was also commissioned as a major. This group of workers made a preliminary survey of the disease in the Middle East and Palestine and set up an experimental ward and laboratory as part of the 38th general hospital at Camp Russell Hustedt, in the desert, approximately eight miles outside of Cairo. One building was especially altered for this purpose and screened with special care against sand flies. It was ready for use on 20 May 1943. Apparently we had arrived much earlier and was maintained as an active laboratory until 15 December 1943.
Now the objectives of research on these diseases were as follows: as an immediate step to determine whether any of the mosquitoes repellents—because there was no specific sand fly repellents—might be effective in protecting against the bites of the phlebotomous pochetassay, a procedure which might then be used in an attempt to protect against this disease. Secondly, to recover one or more strains of the virus of sand fly fever by reproduction of the disease in human volunteers, and to make positive identification by transmission through phlebotomous pochetassay, raised in the laboratory from ova flies previously proved to be non-infected. This is very important. So you see this man going out, catching the sand flies, having them feed on volunteers, make sure they didn't transmit and have them lay their ova, and have it raised. You see, it was a complex operation.

To attempt to infect a large variety of lower animals and cultivate the virus in embryonated eggs simultaneously with the work on infection in human volunteers. Fourth to develop an adequate source of virus from lower animals. Embryonated eggs, insects, human beings, which might be used for the elaboration of a specific diagnostic test as well as studies for artificial immunization against the disease. And to invest finally, fifth, to investigate the possibility of transmission by vectors other than phlebotomous pochetassay, especially parasitic arthropods indigenous to
epidemic zones and mosquitoes prevalent in the United States. In all this information you didn't know. You have to get understanding of a situation. We had no way of knowing whether somebody arriving in the United States, let us say in the incubation period of sand fly fever, if you went somewhere in the United States, or somewhere or in another theater of operations that insects other than phlebotomous couldn't then pick it up and then set up a new chain of transmission. A knowledge was really primitive about it.

Q Well, this is why I asked you--

A We were in a search for understanding here at the same time as one tries to prevent it as much as possible. And that is why the first job that I was assigned to on this particular thing--this was my baby together with Cornelius Phillip--was to determine the effectiveness of repellents on the natural conditions. Before I say something about this, I should say that the commission had certain other interests. Now, Dr. Paul had been interested for some time in the question of hepatitis. Of course, hepatitis had arisen first as a big problem in the United States when so many thousands developed the disease after yellow fever vaccination. Then the first experiments of transmission to man that--the establishment of hepatitis as a viral disease was really done during World War Two. So hepatitis was one of the things that followed. And thirdly, since we were there also as polio specialists, so to speak, both
Paul and I were there to keep an eye on poliomyelitis and polio like disease among U.S. and allied forces in the area. Now the division of labor was that Dr. Paul would be concerned with hepatitis and the study--epidemiologically and otherwise the problem of poliomyelitis. And that Cornelius Phillip and I would have the major objective on phlebotomous fever sand fly--fever and phlebotomy.

Well as it so happened on our adventurous trip from the United States to Cairo, Dr. Phillip, major Phillip, a wonderful person. He was a real character. When we arrived at Attron, he practically fell on the ground and kissed the ground in recollection of the years of work he did there on yellow fever transmitting mosquitoes. He said mosquitoes don't bother me. But when we arrived in that area, the talk of the place was, and there were warnings all over--that the place was full of infected mosquitoes transmitting malaria. Well, Dr. Paul and I tried to heed this. He didn't. He was a great swimmer so the first thing we put our gear down in some barracks to which we were assigned, while waiting. He rushes out for a swim at sundown. You see. Well, he was bitten by a mosquito. That didn't bother him. He came back the next thing was--is this the place here to tell an anecdote.

Q Yes. Please. As a matter of fact, the more anecdotes, you tell me the better.

A Alright. The next thing, we had to wait over night for a plane to take us the following morning across the African belt to Kahrtum. Nothing to do, and they had a G.I.
show. But there were warnings out that said unless you have mosquito boots, don't come to the show because they are biting all over. Well the three of us went to the show. We felt some bites. The first G.I. to come on the stage, the master of ceremonies, starts off the following way. He says, "I want you guys to know that I came out of a sick bed to entertain you. My girl's got the malaria again."

Alright. So, at this stage, scratching ourselves a little bit. John Paul and I decided we better go back to the barracks. The hell with the show. But Cornelius Phillip—Phil as we called him affectionately, said we were a bunch of sissies. He was going to stay. So the following morning, we go, we arrive in Cairo, and then while things are being set up for study, the first thing that had to be done was this study on effectiveness of available mosquito repellents.

The only place at that time May, 1943, where phlebotomide were already available was by the Dead Sea in Palestine. So, we started out. Now, the only people—. Now, although Cornelius Phillip was an excellent entomologist—he knew lots about mosquitoes—he didn't know anything about phlebotomide. Because he had never seen one. He had never worked with any. We don't have them in the United States, except a few species in Okey Fenocki Swamps in Florida. So our contacts were the laboratory of the Hebrew University in Mount Miscopis, laboratory of parasitology, under the direction of Professor Adler who had been using phlebotomide
to study the transmission of lice moniasis. And they were experts on phlebotomous flies. And besides, the British forces there also had some cream which they would like to have tested. We decided to compare American mosquito repellents and British cream repellent, and we decided that the only place to do it was to go to the Dead Sea, the Pottish Works, and to take American volunteers—soldier volunteers with us, and to test it out there. Where we would catch the sand flies. Well, on our way from Cairo to Palestine, basically we were on our way to Jerusalem. But we had to stop—there was an American Hospital in Tel Aviv at the time. It was called The Tel Lajinski Hospital. And this jeep ride from Cairo across the Sinai desert, to Tel Aviv was achieved in something like eight hours. We went lickety-slip over those roads. Some of them covered by sand, and tar melted. In a military command car. We discovered the reason the sargeant was driving this way, was driving us this way was that he had a date in Tel Aviv. He wanted to get there as fast as possible. Well, he just shook the hell out of Phil and me. And when we arrived and we had a blow-out in the desert and we had to change it. Finally we arrive and a little late we get to Tel Lajinski. The commander, American colonel and commanding the hospital said "What took you guys so long. I'm sorry I can't stay with you. There is a cocktail party in Tel Aviv. If you want to go with me, wash you face, come with me." So we just washed our face and we go to this cocktail party and
as we are introduced, this is General Motors representative, very polity, and as we passed, they giggled. We wondered what the hell was so funny about us. And it turned out that this horrible ride across the desert wore out holes in the seat of our khaki pants and we--.

Well, at any rate, all this is important. Because there was also a lot of drinking going on that night, and my good friend Phil was a hard drinker. And the following morning, we take off in a military car to go from Tel Aviv up through the Judain hills to Jerusalem. To Mount Scopis. Where we are going to get some guidance in setting up these experiments. Well in the middle of this, my good friend Phil who was full of all kinds of stories and all kinds of help, suddenly began to talk in a way that was peculiar even for him. As I looked at him and felt him he appeared that he was hot. I had a feeling he was out of his head. He was just about two weeks since we were in Attron. And I had an awful feeling that he might be coming down with the encephalitic form of malaria. Falcipron malaria. He was completely out of his head, what he was saying. So as we got to Mount Scopis, and the first thing we go up to Professor Adler's laboratory, and I knew Professor Adler, I said to him, I took him aside and I told him what I suspected. But then in front--because Phil would have killed me--I said look, you are experts here I think before we get started on this, just examine a drop of blood from
me and Phil here and see whether we've got anything circulating. You are the great experts here, malaria and other parasitic disease. Well, it turned out that Phil's blood. Major Phillip's blood was teeming with malaria parasites. I immediately took him back to the American hospital in Tel Lajinski, and I knew that here was the enthomologist of our commission knocked out by malaria. When I took him back I felt very sad. But he was in that gay phase. You see, high fever was happy and--take him to the hospital and as we get on the ward, the nurse turns to him and points to me and she says,"Major, what's the matter with your pal there, he looks so depressed and sick."

So, Major Phillip was hospitalized and ordered to come back. Fortunately, there was a young man by the name of Arkin, well trained in phlebotomous. Professor Adler spared him. This was the first example of the role with really the Hebrew University personnel played in British and U.S. military operations in the Mid-East. Because they were the people who were draining the swamps, fighting malaria, every disease you know, that was a barrier to development. This was only 1943. And they were really well-trained. And they were the source of tremendously important man power for the medical services in the area. And so this man was assigned. He knew a lot about phlebotoma. I began to be trained as an enthomologist. He went down with us. We set up shop in some huts by the Dead Sea and the Pottish Works.
We had a group of American soldiers as volunteers, and we used to go out in the morning. To the white washed huts of the pottish workers to catch the sand flies. It was very easy because on the whitewashed walls they stood out. Because their natural history is they come in, bit the workers at night, they bred in desert areas nearby and rubble and so on. Then after they have a blood meal, they just can't fly. They get so heavy with blood. So they would be on the walls there, and you could spot them. You see. With a flashlight, or even if the sun was shining. You could see them, and them with a suction tube, you would collect them. We had teams you see, to collect thousands of them. And then keep them, and maintain them in the ways that our controllers knew how to do. And one of the interesting sidelines here I remember Dr. Paul arrived by that time, and we had an English lieutenant colonel with us. From the medical services. As we would go around these huts, they were very simple. There was a cot. And by the cot invariably, there was a small stand with books on them. And we would look at those books and what was there. Philosophic works. Collected works of Plato. Shultenhauer. Spinosa. Meacham. And I remember John Paul saying to me, "What the hell is going on here. What kind of people are these? Pottish workers reading Plato, and Shultenhauer, and Meacham. And this of course was a reflection of the intellectual status of the people who were doing manual, of the Jews who were
doing manual labor in Isareal at the time. Many of them were refugees from Germany or from other places who had high professional training but there was no need for their professional training. And they worked on the roads. They worked at the pottish works. Well, at any rate, it was there that we did our tests, phlob--

Q Dr. Sabin, you know, at that time in Israel was an old Rockefeller hand who had worked with Oletsky named Kliggler. Did you ever run into him?

A Kliggler at that time was the chairman of the department of microbiology of the Hebrew University precise on Mount Scopis. I knew him when he had come back to New York prior to the war when I was at the Rockefeller. And of course, Kliggler was there when I arrived--I knew him just as I knew Professor Adler before he--before I met him for the first time. Of course, the interesting thing was, there was also a brand new in--it was also the place where the clinical unit was--the Hadassah Hospital. In an entry--the beautiful entrance, marble hall, there is a marble map of the United States with the different states in Hebrew letters. Of course, the relationships, the impact of American training and British training on a people who were manning, were forming the nucleus, because there was no medical school as such yet. But it was a clinical center, a hospital, a laboratory, that had pastology, microbiology, and so on, were very very great. And Professor Adler himself was born in England and he was one of the great parasitologists in England who came--
he settled in Palestine. Dr. Kliggler came from the United States. He worked, was an extraordinarily good group at the time. We would have been really lost without the help of these trained people.

Q Now, were you able to transmit the--

A Well, of course the first thing were the studies of the repellents, and this was done in several ways. It was done by smearing some repellent on, on parts of the arm, and then at different times putting on a counted number of hungry sand flies, special container to feed on that area and see whether or not they would feed. We'd have them on the control area of the same individual. Because there are individual differences. So you would have them feed on an untreated area of skin and on a treated area of skin. And in that way determine the relative effectiveness of different mosquito repellents. The American repellents were fluid. There was a British cream. Well, after all of these studies gave certain indications, then we had presumable tests--tests on the presumably field conditions. In which just before it became dark, we would let a certain number of volunteers cover themselves with the repellent because it is hot and you had to sleep with very little on. And large surfaces are exposed. And we would have others who were not. You see. And we would just have them in the room and it would be dark. And then I would come in and release a thousand or a couple of thousand hungry phlebotomide.
And then I would couch the ouches. Or I would have each man sort of make a record who was bitten and who was not, and how much and then we obtained evidence in that way that those were really--because it was a matter they had to do it on their hour inspection. I mean, here is a bottle. Put it on. With those who really had applied the repellents, were protected to a very large extent, whereas those who were not, under the same conditions, in the same room, with the same air circulating through it, and all that, were not protected. So the job was done, and the recommendation went out we had the best American repellent--that came out of that.

Q Dimethol--

A Dimetholtylate. Yes. And basically the recommendation was that this be used. Subsequently we had occasion to test it in Cairo where military forces were. So that part of the job was over. Then came the job of what is sand fly fever. And how do we get some of the sand fly fever virus. And then also, a volunteers. Our volunteers had to be American soldiers. We had to get permission of the military to let a certain number of soldiers come, be quaranteened, and then we get blood from them. And all sorts of things. Work them up, in this special unit. Investigational unit. So then we had to have the material. From the field. And, so there were certain criteria for diagnosing phlobotomous fever, which was something that would produce a illness. Pain in the eyes. General malaise. It would not
last more than three days. Self limited. You see we wouldn't use it. We would take blood from them at the onset and then freeze it down, you see, because we had refrigerators with dry ice. We would keep the blood in. And then when it turned out that they didn't develop any other illness, and of course, we cultured it from the blood and what not--try to eliminate other things. But of course that doesn't--it is all circumstantial, and if there were biting sand flies in the area, the presumptive diagnosis of sand fly fever was--. And it was this stuff that we would then inoculate in soldier volunteers to see what would happen.

Q In other words, there were no animals that you could give this to.

A No, because there were no susceptible animals. I mean, animals were inoculated to make sure that it didn't have anything else, just like cultures were made of this stuff. And sure enough, within a given period, incubation period, the volunteers who were inoculated developed the fever, that was similar and reproduced all the manifestations. Now to prove that this was actually virus disease, it was necessary to then take blood from them at the proper time and give it to another set of volunteers to see if it would reproduce it the same way. And it did. We went through all of this. And from then we bled. And every volunteer was bled rather extensively. We put the stuff away frozen in dry ice, and now we had our source material. But then that wasn't enough. We merely proved that we had something
that was transmissible, and through filters and what-not, bacteria free. But was it phlebotomus fever? In order to prove that it was phlebotomus fever we would have to prove that sand flies, phlebotomus flies that were uninfected but we would hatch out from eggs, you see, so we had an insectory set up, that would hatch up from eggs, and which by themselves were uninfected. So we had to put them on to feed on certain volunteers beforehand to make sure that they didn't infect anybody. That when they were then put on to feed shortly after onset of fever, and they were allowed to go through a period of digesting this blood, I forget now what the period was. That when these sand flies were put on to feed on other volunteers that they would reproduce the disease, on whom in turn would we could put other sand flies on to transmit in turn and each serum would—in other words, we had to do the whole thing from scratch in order to prove that it was sand fly fever. We felt we could do it that way because out of the whole group, all of us were tested because I had been tested and developed sensitivity, incidentally, to bites of sand flies. None of us became sick in those tests that were done with the repellents. And it was from those sand flies that we also had collected eggs to hatch out in the laboratory. We didn't use wild sand flies for this. We used the eggs that were collected. You see we had to be trained by this guy Arkin. This again, where, and this carried over.
Because, again, without the help of the Hebrew University people we would have been lost. During all of this period, our good friend Phil was in the hospital.

So we had quite an insectory there and all of this work was done. It was established—. But then of course, we would always go out in the field and get more—field which was on the Suez was a rich source of this. And interestingly enough, subsequently, we also had specimens from Sicily and it turned out that we did experiments—

Q Did you go to Sicily?
A Well, this is another chapter. At any rate, many of the things were learned during this period of real, chemical, laboratory investigation. Of course, we tested the possibility of propagation of eggs which was erroneously reported as from India. I mean there are a great many details which I won't go into.

Q I don't want you to—.
A Alright. So the point was incidentally, and among other things, that in trying to determine whether serum containing the virus after inactivation and heating it 56° inactivated this virus, would produce skin test and people who were immune to what it might do to people who were not immune. So we used ourselves in the laboratory as well as some of our soldier volunteers. And as it turned out, while heating at 56° centigrade, destroyed the sand fly fever virus, it didn't destroy the contaminating hepatitis
virus that happened to be there. We got it from people in the field, you see. There were eight people including Dr. Paul, Captain Havens, who was assigned to us—Paul Havens, myself, and five others. Well, out of the eight people who had the skin test done on them with 56° centigrade inactivated serum, three including Dr. Paul, Paul Havens and one of our soldier volunteers developed real, severe hepatitis. And even though Dr. Paul had had a history of having hepatitis before, this was another attack. Of course, at that time we didn't know about different types of hepatitis. And it actually caught him—because the incubation period was something like two months—he was on a mission somewhere in North Africa. At any rate, this led them and Paul Havens into the field of study of hepatitis and that is the subject of a paper that Dr. Paul reported it as transmission of hepatitis.

Q  Yes.

A  This was my first venture into giving hepatitis to my colleagues, without knowing it.

Q  Well, you have now spoken of the diseases that Paul acquired and that Phillip acquired. But you too got sick.

A  Yes, well this was another story. During the course of this work, as I said, we had assignments other than sand fly fever. Now, Dr. Paul was trying to get monkeys from different parts of Africa. And some of them were real tough. There were being flown in from Ethiopia for various experiments
which we would use for hepatitis, polio, whatever we suspected. And I remember another anecdote. Unlike the rhesus monkeys, some of them we got from India. These African monkeys behaved quite differently when you tried to catch them. In the procedure at that time was, we didn't have the various types of cages and in which you could hold them down and catch them. The procedure was that stuff that I grew up on as a polio virologist, and Paul grew up on. We would let the monkeys out of a cage and in an enclosure, they'd run around, you see, on wire fencing and in the first place, you would be able to detect any paralysis very nicely that way, and that was the way you caught them, while they were on the wire fencing. But while rhesus monkeys always presented their back, you see, their face was against you. These African monkeys turned around and if you would try to catch them they would come at you. We had some G.I. helpers and I remember some of them going in there, to catch them. And John Paul was giving them directions from the outside what to do. Well they were being attacked. These monkeys would come at them, they finally came out and they said, "Dr. Paul, we are going to ask for a transfer. We'd rather be out fighting than facing these monkeys." So Dr. Paul said, "Let me show you." Now he was small, physically small--Dr. Paul going in there. And by God he felt it incumbent on him to do what these guys couldn't do. He had never faced an African monkey before. And by God, really the courage he displayed--
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and the way he--he was a small person--he didn't give up, you see, until he caught some.

Well, so now let's go on to this point. That since paralytic manifestation of polio was one of the problems that we were concerned with, there was a disease that while it wasn't too common, it wasn't claiming many, it was diagnosed as brachial neuritis. It would involve the arms, and shoulders, and paralysis, and of the other signs. And one of the questions was--it wasn't clear whether it was an infectious disease or not. And one of the questions at that stage of our understanding of polio was whether this could not be a form of polio virus causing this type of paralysis, especially as selected in the children very often. We had a report of two American soldiers with this disease in the American hospital in Tel Aviv. So we asked to have stool specimens and blood specimens sent. And in those days, the way we used to handle stool specimens in the United States and elsewhere to prevent overgrowth of bacteria would be to have ice cream cartons, and the patient would defecate into it. And in order to keep down multiplication until it got to you, you would put some ordinary anesthetic ether in because anesthetic ether didn't hurt the virus. As a matter of fact, this is the way we destroy the bacteria and got the virus. So this was done, and caps were put on these ice cream cartons. They were sealed. And the idea was that they were to be
sent by plane just as quickly as possible from Tel Aviv to Cairo and brought to our lab. The temperature in that plane must have been well over 100°. Finally it comes to the lab, and this was my job. You see, I was the man with the polio experience. I had already done thousands of monkeys. Paul was doing other things. And I get these cartons in with the stools from the patients with brachial neuritis. And as I open it, the ether had been under such pressure there that the stuff in there, you see, just poof went up right in my face. I didn't think any more of it. I corralled up the stool specimens and inoculated some monkeys that we had. Watch the monkeys, watch the monkeys, nothing happened to them. No polio virus. Ten days later I developed brachial neuritis. And I was hospitalized in this 38th general hospital outside of Cairo. I became interested myself in what could have happened--I mean, the connection was very close after all. There was no other case of brachial neuritis. So I probably got it from--. Well, the only thing that came out--and this left an impression on me was that apparently I must have picked up a paratyphoid salmonella infection from this explosion on me because I had this salmonella in the stools. And, the only hypothesis that I could come up with was that like certain other things, I studied before that Landragge run by Ray's syndrome, and other things that the probability that the brachial neuritis syndrome was a manifestation of an infection with certain enteric organisms that liberated a toxin with a
special affinity for the nerves of the brachial plexus. I couldn't prove it. But this remains in my mind as a mechanism for the production of different types of nervous, particularly periforal nervous system, and perhaps even some central nervous systems manifestations caused by toxins with special affinities. Some for the periforal portion of the neurons, some for the neuron itself. Some for nerves in various parts of the body. Some selecting very specifically the brachial plexus. But that was an interlude that put me in the hospital for a while without contributing very much except that in these two cases, an American soldier certainly there was no polio virus that you could isolate in monkeys.

Q You know, the British troops in the Mid East had a very high incidence of polio at this time. Far higher, for example, than the native population.

A Go ahead.

Q One of the interesting British virologists who was out there was a man by the name of Van Royan. I wondered whether you had run into Van Royan or not.

A Oh, we had lots of contact with Van Royan. I first got to know Van Royan when I worked at the Lister Institute in London in 1934. And you can imagine how surprised when we arrived in Cairo to find the good old man as we called him, was at the Scottish Hospital in Cairo. Well, Van was always exploding with all kinds of ideas. And very often, he would call us at midnight and ask for John Paul or me,
and he'd keep talking. Say for God's sake Van, what is there in this particular telephone call that couldn't have kept on ice or off ice until tomorrow morning. But at any rate, we had a very nice relationship with Van Royan. But, this was one of the things that John Paul undertook to study. During his stay there, he tried to find out just how much there really was in the native population. And he studied hospital records. He went around every where. The general statement was oh, there is no polio in Egypt. But when he went around to orthopedic clinics where the consequences of polio were studied it was obvious that there was. I mean, not a great deal, but there was polio. And one of the reasons perhaps that it wasn't recognized early in the acute phase is that polio infection in Egypt or under conditions obtaining there, it is transmitted so early in life, before the children begin to walk, that unless the paralysis is very extensive, so the child doesn't kick or do anything any more, you don't find out until the child has to begin to walk or to do things. So that, most of it, you see, is actually is because polio clinically is a spectrum. Most of it was actually seen after the children got old enough. So, first of all, Dr. Paul established that of course, there was polio. It was occurring very early in life. Quantitatively there was no way of estimating how much. And there was also no question that in British and American troops stationed in the Middle East and the Far East and India and other places
polio was occurring out of proportion to what it was occurring let's say in the United States in the same groups and certainly what was occurring in the population. This was one of the things that influenced our subsequent thinking contributed to the ultimate putting together the pieces in the puzzle of the natural history of poliomyelitis that among the strain of polio that were being disseminated in certain areas, that people coming in from the outside, particularly young people such as those serving in the army who were not immune had a much greater chance of picking up viral and polio virus than if they remained at home. But the only tools that we had available at the time was to inoculate monkeys and to prove that polio was polio and brachial neuritis was not polio. And aside from clinical epidemiological observations on poliomyelitis in allied forces, there wasn't much else really that could be done.

Q The reason I raise the question about Van Royen is that some serum was taken from British soldiers in the Mid-East and sent to the Rockefeller Institute. And Dr. Oletsky's laboratory established the virus as a Lansing type II virus.

A Of course this was done in a number of different places at that time. I remember that 1939, Dr. Armstrong adapted Type II. Polio virus as--adapted type II, a type II polio virus strain in mice. And there was a mouse that had that strain in which you could do polio. What it really
was that in some of the patients, there may have been a rise but certainly it wasn't all type II polio. Predominant type undoubtedly was type I and type III which they never would have detected. And merely demonstrating antibodies meant nothing because these soldiers could have had type II antibody before they went in and a slight rise as our subsequent studies showed, could be a non-specific rise, and since there was no potential at the time of measuring type I and type III, those results really meant nothing.

I am sorry to say that they didn't show anything, and they only show that some of the soldiers had antibodies for type II, so that the vast majority of the population of the United States, it was an early stage of using the mouse-adapted virus, and the demonstration of a slight rise, or a rise in antibody merely showed that in some instances, there was a rise but it didn't necessarily mean that it—the polio that caused the paralysis was the polio responsible for the rise. That, later on, in my later studies, I demonstrated that you can get group related rises. That when you are infected with one strain, with one type of polio virus, and you had a previous infection of another, that when the antibody was the infecting type, goes up, the other one may rise transitorily. These are studies that were done later.

Q  Dr. Sabin—
A  There were not too many tools, you see with which to work.
Q  Well this is why I asked the first question.

A  As a matter of fact, we didn't even know at that
time of the existence of three different types. We only
suspected that there were different types. But the actual
program which was carried out later years, after World War II,
which provided us with information that there were three
distinct types was not available at the time.

Q  Were you concerned with different types of sand
fly fever?

A  I was not concerned. It came out as a part of
studies because one of the things that I studied in the
human volunteers was whether or not a single attack provided
immunity to reinfection, let's say weeks or months later.
And the reason for doing that was that historically,
people in Palestine. I mean, many of the Jews who had come
and worked there during that period and elsewhere. There
were stories that oh, yes, I had an attack of sand fly fever,
but then I had another one, and another one. The issue
of course was a difficult one because there are other
infections and other viruses that we know now--the
enteric viruses, the echo posackies
and others can produce infections which can simulate this.
So there was really no way of knowing whether they had
another attack of sand fly fever or whether they had another.
So we believed it was necessary to study it in volunteers
on the station. And so, in isolating different strains of
virus, we kept them separate. We didn't work with pools.
We studied the resistance of volunteers who had previously gone through a known infection to reinfection. And we could show that they were resistant. Controls inoculated for the first time with the same material came down—it was done in the standard procedure. And with strains that I had obtained—strains means a specimen of one person having the presumable disease somewhere in the Middle East. Not only in Egypt around the Suez Canal but other places. I found that resistance to one also meant resistance to another. But towards the end of my stay in Cairo, the Sicilian campaign was on. And Sicily had been invaded and I received orders to go to Sicily and see what role if any, sand fly fever was playing in the field.

One of the reasons for it was that there was a very large number of people who were being sent back, who were being diagnosed as malaria. One of the mistakes in diagnosis that was suspected but not proved was that the onset of sand fly fever can simulate malaria to such a point, that even if the smear was negative, for malarial parasites, in the field, they started treating everybody with anti-malarial drugs. And take them out of the line, you see, and send them back. Well as the reports were coming in from Sicily, there were quite a number of reports of negative smear malaria. So we became suspicious. Because we were in touch with headquarters at Cairo. So it was arranged for me to go over in the field—and I reported to
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Palermo, got a jeep and went out into the invasions. To get first hand information. I wrote a detailed report on this. I don't know whether it is in here, but it is in some of the documents. And I discovered the following. I discovered that the proportion of malaria--of negative smear malarias was fairly high. I don't remember what it is now. I recall it--

Q That kind of detail we can look up.

A So I felt it necessary for me to go out into the field and to convince some of them medical field officers to when they made a diagnosis or when somebody came in with fevers to let me take blood out of them, and process it and freeze it. And then observe the patients. Not treat them. Some of them, if their smears were negative. I mean, if they had a positive smear and malaria parasites were there, sure go ahead and treat them. But I said, let me see some of those with negative smears and don't give them any treatment for God's sake. I want to see what happens next. And that they ran the usual three day course or so and they got better. Of course, many of them were as a period perhaps some strain and more often than others of tremendous dability. Dability after the temperature goes down. Because I remember the chief medical officer, American medical officer in Palermo at the time saying that he had it and he was knocked out for ten days, two weeks he just couldn't work. He had a terrific depression and everything. So I followed a number, and sure enough the ones I followed, negative smear malarias recovered in three days. Some
were all right. They could go back to work. Others maybe needed a few days. But without any antimalarial therapy, they did all right. By that time I was ready to go back to the United States. To my laboratory in Cincinnati because it was felt that in order to pursue the work further, on sand fly fever, it was not possible to do it in the field. I would have to have larger numbers of volunteers, and to do certain things that I could not do. So basically, I took these samples that I had consistently back with me to the United States along with all of the material that I got collected in the least in Cairo.

I started to work in human subjects in Cincinnati. And sure enough, with the--of course I couldn't do transmissions with phlebotimus there.

Q Now, this is the--
A Muguay.
Q I wish you'd cover that.
A Alright. Because it was absolutely forbidden to import phlebotimus into the United States. So I inoculated human subjects with the material from the soldiers in Sicily and found that it reproduced the disease just like the stuff in Cairo. And then I challenged them to see whether they were resistant to the virus that I got from Sicily and whether once they were resistant to that, they were also resistant to the Middle East strain that I had. And sure enough, they were. I said, that is simple. But as time went on, and by that time I had left Cincinnati and I went to work at the Rockefeller Institute at Princeton
because I needed more human volunteers, and besides the problem of dengue became assigned to me. And then, after the American forces moved up from Sicily and got into Naples, this negative smear malaria or short three day fevers, of unknown origin began to occur in considerable numbers in Naples. And I received specimens that were obtained by well-trained officers, one of them incidentally was Fred Robbins, you see, who later on went to work with Anders and Wella. And I passed some of those sera, of course always after preliminary tests to make sure because there was also Que fever there and there were other things. So those sera kept frozen, aliquets had to be tested out to make sure that there weren't other infectious agents there before it was given to any volunteer. But we inoculated some of the stuff into volunteers and I think this was if my memory serves me correctly, it was first done in the Trenton State Prison because I didn't get those specimens while I was still in Cincinnati, and again there was reproduced a three day fever. Of course I couldn't do a phlebotomous transmissions. However, I determined whether they were resistant to this strain of virus by reinoculating control and so, that business. And then I challenged them with the Sicilian and the Middle East strains. And lo and behold we got another attack. And then I did it the other way, you see. I inoculated volunteers with Sicilian Middle East strains. They got the disease. They repelled, they were resistant to their own, but when I gave them a shot of the Naples, they were not resistant at all.
So here was the first evidence obtained entirely by human as my studies on human subjects that there indeed were two different immunologic types of the virus. Of course other studies then were done by determining the size, the filtration and other properties which show that it was the same. And then also we undertook our first studies at adaptation of the phlebotomous fever viruses to mice. Of course all this was established in this part of the archives of viruses that are available so that now there are two types. One is called the Sicilian strain and the other is called the Naples strain and they are both sand fly fever but they are immunologically totally different up to the present time we have no evidence that more than two types but one cannot be absolutely certain. One of the things that happened very quickly during that period was that when DDT really came along, towards the latter part it turned out that DDT was more effective against the breeding places of phlebotomous flies than almost against mosquitoes and therefore, DDT by itself served as an excellent way of controlling the spread of sand fly fever. I think there is another footnote if I may add here.

From my observations in Sicily. There was the place within which all the knowledge accumulated by years of research on repellents, mosquito repellents. Our own research on the effectiveness of these repellents against phlebotomous flies. Our own research in the field tests
that were done to show that it really was effective on the natural conditions. It could have been applied the ware houses in Sicily I discovered were full of these repellents. But they were not used. Why were they not used there? Because I went around with the jeep in the field and all that. Couldn't you get any? Where was the Snafoo? Oh that stuff is kind of greasy a soldiers don't like to apply them. Besides, in the heat of battle how the hell can we stop and--. Well, the important point here is this. That in effect, the purpose for which these repellents had been developed, all the research was done. When it came down to the time of utilizing this knowledge, where it was needed, the last step was completely overlooked, namely what would be the requirements of having this used in the field to protect against mosquito bites to protect against phlebotomous fly bites. To protect against those insect transmitted diseases in the army, and it was a necessity of having a special relationship established with command whereby you would almost have to have the command officer or lieutenant or the captain or the sargeant, as a routine when sundown comes, everybody lines up and puts on this repellent. You see. Under orders. Not given a bottle, use it or not. The precise technique of use, at the end point it was made for the man, for the soldier. But that last step was not taken care of and all our work was for nothing. From the point of view of doing, having a
beneficial effect on mosquito and phlebotomous transmission diseases in the army. I learned an important lesson. And I am not at all sure that the army has as yet learned that lesson. They learned that lesson in part, yes. When addabrin was shown to be so effective as a prophylactic agent for malaria and in South Asia, malaria continued to occur anyway. I think that MacArthur really got sore and told the men God damn it this malaria's got to stop. You've got to do something about it. And considerable effectiveness of addabrin was obtained by having them swallow that, to watch them swallow. To have somebody see that the man who is giving the tablets swallowed it, down. So he couldn't hide it. It takes that sort of discipline and making sure that a whole lot of research is properly applied in the field.

Q  Dr. Sabin, we are almost off the tape so I am going to --

END OF TAPE