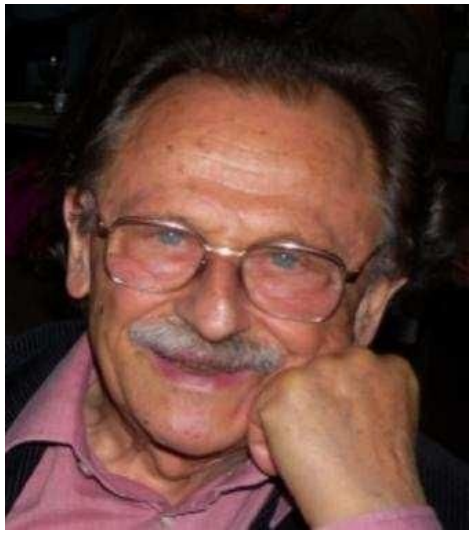
Jean Claude Kaplan



Personal Details	
Name	Jean Claude Kaplan
Date of birth	21.09.1930
Place of Birth	Paris
Main work places	Paris
Principal field of work	Inherited metabolic and muscle diseases
Short biography	See below

Interview	
Recorded interview made	Yes
Interviewer	Peter Harper
Date of Interview	19.04.2005
Edited transcript available	See below

Biography

Jean Claude Kaplan was born in Paris in 1930 and studied medicine and biological sciences at University of Paris. After residency posts in internal medicine and paediatrics he joined the laboratory of George Schapira at Hôpital Necker-Enfants Malades, Paris, in 1962, for research on inherited metabolic disorders. In 1967-8 he was a Fulbright Fellow with Ernest Beutler at Duarte, California, and from 1969 became Professor of biochemistry, and subsequently of molecular genetics, at Salpêtrière Hospital and then at

Hôpital Cochin, Paris. His principal research has been in the molecular basis on inherited muscle disease.

Interview with Professor Jean-Claude Kaplan, 19th April, 2005

PSH. It is Tuesday 19 April and I am talking with Professor Jean-Claude Kaplan at Hôpital Necker, Paris. Jean-Claude, you have written in the interview that you gave with Dr Picard about how you became interested in medicine and the early years, but I would like to ask you first at what stage did you become familiar with any form of genetics?

JCK. Rather late in my cursus. I thought you were going to ask me at what age I became interested in medicine, a question to which I have a ready answer.

PSH. Well perhaps you can answer that first.

JCK. The answer has to do with heredity. I wanted to become a doctor quite early, at the age of 4 or 5, because I was homozygous for the trait, since my parents were both doctors; coming back to your question about genetics, I must say that I never got a basic training in genetics as a specialty. Primarily I studied medicine and biochemistry. During 4 years of clinical residency, mostly pediatrics, I became more and more interested in the analysis of the biochemical basis of diseases, to such a point that, after obtaining my MD degree, I deliberately left the clinics for the bench. This choice was also determined by a short experience of private practice. It made me realize that I was more suited to study diseases than to take care of patients.

I joined George Schapira's lab in 1961. He was Professor of "Chimie Pathologique", and head of a lab of biochemistry at the Hôpital Necker Enfants-Malades. He wanted to attract young MD's specially trained in biochemistry to develop the novel field of molecular pathology applied to human inherited diseases. My interest in molecular genetics developed later, when genes became biochemical objects. This was around 1978. In the meantime I had been involved in biochemical research on inherited protein defects and particularly in human red-cell enzymopathies.

PSH. May I ask, was Professor Schapira at that point at this hospital, or was he at Cochin?

JCK. No, he had started at the Hôpital Necker-Enfants-Malades just after the war, when Professor Robert Debré, a prominent figure among French paediatricians, asked him to create and develop a service of biological investigation dedicated to paediatric diseases. At the beginning the space and facilities were scarce. The group expanded progressively, and in 1968 it moved to Hopital Cochin in a new and big lab with much better facilities. It became later the Institute of Molecular Pathology.

PSH. And was his service part of the overall children's service with Debré or

was it a separate laboratory?

JCK. It was a separate laboratory. Robert Debré always supported Schapira, but he never tried to "phagocyte" him. From the beginning Schapira's lab was independent from the paediatric unit.

PSH. That's very interesting. May I ask then, you also at that time were working here at Necker?

JCK. I joined Schapira's group in Necker-Enfants-Malades in 1961, and I followed the translocation to Cochin seven years later.

PSH. At that time, was there any close link between the group of Schapira and people such as Lamy, Jean Frézal and others?

JCK. The only links were geographic, everybody being in the same hospital. But in terms of research, there were no links at all, instead there was a real gap. This is because George Schapira with his double background in medicine and basic science had a far-sighted vision of the future of medical sciences. He strongly believed in the molecular approach. This contrasted with the medical community in France in the post-war period still sticking to its clinical tradition. Later in the 50's the French medical geneticists focused on cytogenetics with the success you know. I remember that Jean de Grouchy attended Schapira's staff meetings.

PSH. It seems from what you say and also in your previous interview, that Schapira must have been a quite unique person, at least in France

JCK. Definitely, he was a visionary, a pioneering figure in our country, because he and his close collaborators: Fanny Schapira (his wife), Jean-Claude Dreyfus, and Jacques Kruh had a double curriculum: medicine and basic sciences. They were inspired by Linus Pauling's new concept of molecular pathology. In the middle of the 50's they were joined by other MD research scientists, such as Dominique Labie and Jean Rosa. Everybody in the group used to think and search at the molecular level. This is why they were interested in haemoglobinopathies and enzymopathies. When I joined the group in 1961, people were elated by three recent major breakthroughs: the publication of Jacob & Monod's model of prokaryote gene regulation (lactose operon), the discovery of messenger RNA, the deciphering of the first codon (UUU = phenylalanine). This excitement was not shared by the other biochemists affiliated to medical schools. I was very lucky to join Schapira's group precisely at that time

Coming back to the great figures of that time, you must realize that both Robert Debré and George Schapira were men of exception. The former founded the modern school of paediatrics in France. The latter, with his alter ego Jean-Claude Dreyfus, created the French school of molecular pathology

in medicine. Schapira and his followers were the only ones, in the research hospital community, to be in close contact with the leading groups in molecular biology, at the Pasteur Institute (Jacques Monod, Francois Jacob, André Lwoff, Elie Wollman, François Gros), and at Gif (Piotr Slonimski). Also they had connexions abroad with prominent scientists such as Max Perutz, David Rittenberg, Irving London ...

PSH. I'm very interested to hear you say that Schapira was almost the only link between the mammalian and human biochemical molecular workers and Monod and Jacob, because to me it's extraordinary, that Monod and Jacob and also Ephrussi were so much ahead, and yet in France very isolated almost. I would have expected, because Ephrussi, Monod and Jacob were in Paris that all the rest of people in Paris would immediately go to them and try to form links, but was this the case or do you think they were not understood by most people?

JCK. Such links were an exception in medical circles. You must realize that during these post-war years, and even until the late 60's, the old-fashioned antagonism between medical schools and science schools still prevailed. Both communities ignored each other, and the MD and PhD simultaneous cursus was almost impossible. I experienced that myself.

PSH. Did you go to the courses of Ephrussi and L'Héritier?

JCK. No. These prominent figures were basic geneticists and, as I told you, my academic training was in medicine and biochemistry, not genetics. I am a medical biochemist who jumped on the analysis of defective human genes as soon as it became feasible, i.e. around the beginning of the 80's.

PSH. So when you came to work with Schapira, was Dreyfus also there at that point?

JCK. Yes. He was with him from the very beginning, and he may be considered as the co-founder of the group.

PSH. And what was your first major project then, when you started work with the group?

JCK. Jean-Claude Dreyfus, who was my mentor, was interested in G6PD deficiency, one of the first enzymopathies to be discovered (1956). He was really thinking as a molecular geneticist, and was very excited about Mary Lyon's theory of random X-inactivation in females. I am saying "theory" because her concept was just emerging and was still not universally accepted. G6PD deficiency, an X-linked disease, was a good model to try to obtain a direct demonstration of chromosome X mosaicism in humans. We devised a system to visualize this mosaicism in red cells of female carriers of the G6PD trait. It was a functional test based on the NADPH dependent capacity of

reduction of methaemoglobin. Since the only source of NADPH in mature circulating red cells is G6PD, in the case of mosaicism one should see a double population of red cells: metHb positive (G6PD-) and metHb negative (G6PD+). It is what we obtained through a collaboration with Marcel Bessis, a great cyto-haematologist, who could perform spectrophotometric analysis in a single red cell. Just before us Fialkow in the US, using a different method, had come to the same conclusion, and our work was only published in our vernacular Comptes-rendus de l'Académie des Sciences.

PSH. Was it around this time that you spent your year in America?

JCK. Yes, this was in 1966-1967, in Ernie Beutler's lab at the National City of Hope Medical Center in Duarte, few miles east of Pasadena. At that time I was focusing on red cell enzyme defects and I was fascinated by his contribution to the field. Once, in 1965, Ernie came to visit Schapira's lab and we immediately got along very well. He offered me to come to his place for a year or so, which I did the following year. It was the best period of my life. Beyond the pleasure to enjoy California, with my wife and our two daughters, I discovered the American way of doing medical science.
I learned a lot from Ernie, and notably how to write a paper in English. As soon as experiments were conclusive, he used to jump on his dictating machine and produce a superb crystal-clear manuscript, not a draft. While in Ernie Beutler's group I had my first papers in good journals. All were related to red-cell enzyme defects, with particular focus on the quantitative and qualitative analysis of defective alleles.

When I came back to Paris I concentrated with Alena Leroux on genetically determined methaemoglobin reductase deficiency, and we defined the molecular basis of the two types: type I benign, affecting only red cells and easily managed by methylene blue; type II, with severe neurological impairment and death in infancy. We also developed a prenatal enzymatic test for type II disease.

You can see that the kind of work I was doing at that time, although orientated towards genetic diseases, was done at the protein/enzyme level, using the conventional methodology of biochemistry.

PSH. Can I ask at that time, was the Schapira group still involved with muscle disease or had it finished its time with muscle disease?

JCK. This is an important issue. When I joined the group in '61, the muscle topic had already reached its acme. I am referring to the fact that the Schapira's (George and Fanny) and Jean-Claude Dreyfus were the first to discover a significant rise of serum level of glycolytic enzymes in myopathic patients. They studied mainly aldolase and LDH (late 1950's). Soon after, Ebashi in Japan found the elevation of creatine-phosphokinase (CPK), an enzyme more specific to muscle. Anyhow either enzymes were elevated in the

serum of all patients with DMD and also in female carriers of this X-linked disease. These findings elicited a lot of excitation because they were the first biological hallmark in DMD, a deadly disease that had suffered from about 100 years of ignorance and neglect. Also it raised the hope that high serum CPK might be used for carrier detection, but it was rapidly found that 1/3 of obligate carriers had normal blood CPK, and that a normal level was not conclusive. They left the field of muscle and concentrated on other topics in which the molecular connotation was more pregnant.

PSH. Do you think it was a conscious decision of Schapira to stop working on myopathies because it was too complex a model, or was it that some other work assumed more importance?

JCK. For both reasons I think. It was a courageous and shrewd decision, taken after years spent to study muscle proteins in normal and pathological conditions. Schapira and his senior collaborators realized that the molecular basis of DMD could not be discovered by the then available tools of protein and enzyme biochemistry. They concentrated on diseases more accessible to a molecular approach, such as hemoglobinopathies (with Jean Rosa and Dominique Labie), enzymopathies (Jean-Claude Dreyfus) and cancer and regeneration in connection with isoenzymes (Fanny Schapira). In the lab Jacques Kruh was a genuine molecular biologist. He led the only group working on fundamental subjects, such as protein synthesis, chromatin proteins.

PSH. When did you first meet Bob Williamson? Was this in relation to the haemoglobin work?

JCK. Yes. I think that Schapira and his collaborators met Bob at the Pasteur Institute where they paid frequent visits to Jacques Monod's group. At that time (1961), François Gros had just provided a strong experimental evidence for the existence of messenger RNA as an intermediate molecule between DNA and proteins. Schapira and his team were trying to apply to *H. sapiens* the molecular biology methods and concepts elaborated in lower organisms. They showed that an acellular extract of human reticulocytes could monitor the synthesis of human hemoglobin on rabbit ribosomes. In doing this interspecies experiment, Schapira was inspired by the famous saying of Jacques Monod "*Everything which is true for E.coli is true for the elephant!"* Unfortunately these results were not published in a big journal, and the credit of this demonstration remains restricted to a small circle. Bob was working at the Pasteur Institute in these days and was aware of Schapira's program. Recently, almost 50 years later, Bob told me that he is still impressed by Schapira's contribution in the early applications of molecular biology to human models. He said "*You know, Jean Claude, after all George Schapira was the first to provide an experimental evidence for the existence of messenger RNA in mammals".* Bob was fond of France and came as often as possible. With years we became close friends, and when in 1978 I decided to shift from proteins to genes he was very supportive. I had suddenly realized that

genetics had become molecular, and that the gene approach of inherited diseases opened fantastic perspectives. I was a layman in the field, and had never touched DNA, whereas Bob belonged to the small and exclusive circle of experts in “genetic engineering”. He strongly encouraged me, providing guidance and training.

PSH. May I ask you what triggered your big move?

JCK. I had been fascinated by YW Kan’s presentation at the International Congress of Haematology in Paris showing a Southern blot on which the alpha-globin gene was clearly missing in a DNA sample from a newborn with hydrops foetalis due to alpha-thalassaemia. I was flabbergasted by this first tangible visualization of a gene deletion.

PSH. What year was that?

JCK. The epiphany occurred in 1978. Following that revelation I had to operate a total reconversion of myself and of my group. . Fortunately at the Institute of Molecular Pathology the other group leaders, Georges and Fanny Schapira, Jean-Claude Dreyfus, Jacques Kruh, Dominique Labie, and the young bright rising star Axel Kahn, were also keen to embark into genetic engineering. At the same time at Créteil University Hospital Jean Rosa, a product of Schapira’s school, started working on genes of hematologic interest. The INSERM fully supported me in the building of a P2 laboratory. The big controversy about the presumed dangers of gene manipulations was over, but the regulation about confinement was still very strict. The INSERM also welcomed my first project on human chromosome 22 mapping. Bob Williamson was instrumental in the reconversion. He put me in relation with Brian Young who was performing chromosome sorting in Glasgow. He also sent over for 6 months Rob Elles to teach us the methodology in situ. He also offered to take me to his lab at St Mary’s for some training, and I spent a “sabbatical” fortnight there to learn Southern blotting. At St Mary’s, Bob was chasing the first disease genes with the “reverse genetics” approach, later recoined “positional cloning”. In a sense we were on convergent paths: Bob going from basic science to medicine, and myself the other way around.

PSH. Kay Davies was there at that time also?

JCK. Kay Davies was there doing her post-doc, and searching for the DMD gene. The strategy was to use RFLP’s of the X chromosome to do linkage analysis. I would like to mention an anecdote. I remember Bob in his small office discussing with Jim Gusella and Kay. Jim was after the Huntington disease gene, not even mapped yet. He said he had in his fridge about a dozen of probes for various mapped RFLP. Fortunately among them was the famous G8 probe which allowed him to provide in 1983 the first assignment of the HD locus to the tip of chromosome 4 short arm. This unbelievable luck was followed by 10 years of hard labour to reach the *HD* gene.

PSH. Now chromosome 22, was it at that point you became involved with the gene mapping meetings?

JCK. Yes.

PSH. And remind me, which year was the gene mapping meeting here in Paris.

JCK. That was ’87, Human Gene Mapping 9. It was organised by Jean Frézal who was leading the French school of medical geneticists. Among many other things he was interested in chromosome mapping of human genes, and he was a pioneer in France in establishing with Nguyen-Van-Cong and Dominique Weil the methodology of human/rodent somatic hybrids. The first markers were proteins, and later DNA probes. Jean Frézal chaired the HGM9 international meeting and at this occasion he created the computerized disease oriented human gene database “Genatlas”, which thrived in spite of a harsh competition, and is vivid today, 20 years later, under Martine Le Merrer’s supervision

PSH. I think you were Chair of the Chromosome 22 Committee.

JCK. Yes. At that time this Committee had also to deal with chromosomes 21, and 20.
I was appointed for the first time in 1985 at the HGM8 meeting in Helsinki, chaired by Albert de la Chapelle.

PSH. Did you know Albert before?

JCK. Yes. In 1982 he spent a sabbatical year in my group at Cochin. He was already a renowned cytogeneticist and he wanted to acquire some skill in the new methodology of molecular genetic methods. It is André and Joelle Boué who had recommended him to come to our lab to get trained. I was amazed since we were ourselves almost freshmen in the field! But the Boués’ knew my efforts to develop the new technology in a medical environment and they trusted me. At that moment I was working on a rare non-random translocation variant t (8q24; 22q11) in Burkitt lymphoma cells. In this rearrangement the pathological event was supposedly an undue juxtaposition of the *MYC* gene (at 8q24) and the lambda light chain immunoglobulin gene (*IGLC* at 22q11). This was an ideal model to merge cytogenetics and molecular genetics, and Albert came in quite timely. He succeeded in using for the first time (for him and us) the chromosome in situ-hybridization using cloned probes, and could demonstrate that sequences of the C-region were translocated onto chromosome 8. Two years later Albert was the Chairman of the 8th Human Gene Symposium in Helsinki and he invited me to chair, along with Patricia Tippett, the chromosomes 20, 21 and 22 Committee. I did the same in subsequent HGM Symposia: in Paris with Ben Carritt (1987), at

Yale with Beverly Emanuel (1988 and 1989). You remember that the last meeting of the HGM series, initiated by Victor McKusick in 1973, was held in London in 1991. With Bev Emanuel I organized in 1991 in Paris the first international workshop on human chromosome 22.

PSH. How did it happen then that you came back to muscle disease?

JCK. You remember that in 1961 Georges Schapira had given up the quest for the molecular basis of DMD. Twenty years later D. Botstein, R. White, M. Skolnick et R. W. Davis in their seminal paper explained the fundamentals of reverse genetics, using the newly discovered RFLP as tools for linkage analysis. Remarkably the authors were basic geneticists working on animal models and not on human diseases. However they envisioned a mapping strategy expected to solve several paradigmatic irking medical enigmas such as cystic fibrosis, Duchenne Muscular Dystrophy, Huntington disease. I was illuminated by this paper and I thought that perhaps I should try to fulfil Schapira's dream. Bob Williamson had already started hunting for the « culprit gene » of DMD. He was also chasing other monogenic disease genes : CF and later Friedreich ataxia. He convinced me that this new approach of medical enigmas would soon enter in the scope of academic hospitals. I concentrated my efforts to build a lab and a team dedicated to the direct application of gene pathology to medicine. I campaigned to convince administrators that we had to introduce molecular biology into hospitals. The targets were the AP/HP (Assistance Publique/Hôpitaux de Paris), the INSERM and also the AFM. All responded favourably to my proposal to deal first with molecular diagnostic of DMD. Being new in the field of gene hunting , I did not pretend to participate in the main platoon, but I thought we might anticipate and get prepared to apply the discoveries to patients' DNA.

The project was facilitated by the fact that in Schapira's lab Jean Demos alone had courageously kept busy with DMD, even after this topic was dropped in the group. He was basically a clinician, and had joined Schapira in the early 50s to work on myopathies. He was the only one in the lab (with Raymond Saddi who worked on haemochromatosis) to see patients. He created in the early 60' a specialized consultation of myopathic children in Jean Frézal's department. He used to see many families and in Schapira's lab he tried to find the cause of DMD on blood and muscle samples with biochemical tools. This approach was hopeless and did not result in any scientifically relevant findings. However he also tried to help the families both at the level of common and orthopaedic care, and at the psychological level. He comforted them in the belief that the disease is not helpless and that the search of the cause is a necessity if one wants to do genetic counselling. The AFM has ever since remained grateful to his humane contribution. Thanks to Jean Demos there was a registry of numerous families and a collection of frozen muscle samples. I also approached the AFM (Association Française contre les Myopathies). Its charismatic President, Bernard Barataud, was quite interested when I told him that with the reverse genetics strategy we were «

doomed to win ». This was because in Bob's lab Joanna Murray, Kay Davies, Peter Harper et al had just found evidence that the DMD locus resided at Xp21.2. You remember their memorable paper in Nature (october 1982) reporting the first linkage with the RC8 RFLP, and soon after the paper on the flanking marker L1.28

PSH. Yes I remember very well.

JCK. Shortly after came the big discovery that Becker and Duchenne diseases were in fact allelic. I've forgotten the name of the first author but I remember she was in your service.

PSH. Helen Kingston.

JCK. I was quite excited about this discovery, totally unexpected at least by me.

PSH. I must tell you, as an aside, something about that paper, because as you know until then, it was thought that Duchenne and Becker were at different ends of the chromosome, and when Helen Kingston, who was a clinician but taking a break to do research, she started this work, very diligent and she came to me, with the first family, two families. At that time there were problems with the probes on the long arm and so I said to her well, why don't you start looking at the short arm, then you will be able to exclude it from the short arm and then go to the long arm.

JCK. That's fantastic.

PSH. So then, she came to me and said, I have looked at the short arm probes and I can't find any recombinants. I remember I said to her, well do a few more families because I'm sure what will happen is, the next family you will find lots of recombinants. So she went away and did some more families and still no recombinants.

JCK. This is serendipity.

PSH. That was serendipity. So we showed this and published this, but there was one other thing, we had a site visit from the British Muscular Dystrophy Association, and we had this grant for Duchenne and so we showed them these results on Becker dystrophy and the leader of the visiting group was furious. He said "How dare you spend this money we have given you for Duchenne muscular dystrophy on some completely different disease. Everyone knows it's different!"

JCK. Oh I love that story! But now you are reversing the situation: you are telling me your own story, which I enjoy so much!

PSH. Yes and I must stop.

JCK. Please don't. I like this sort of anecdote. I thought it was some sort of wild guess you made with Bob to check immediately whether Duchenne and Becker were allelic or not.

PSH. It was. Indeed it was, but our hypothesis was to show that they were different, not to show they were the same.

JCK. In fact the concept that they are different was so deeply rooted in minds and books, that even Alan Emery continued for some time to treat the two entities in two different chapters of his book. Regarding genetic diseases becoming molecular I would like to come back to the historical paper of Botstein et al. As I said I was somewhat bewildered that these specialists of yeast or Drosophila would focus their discussion on the expected impact on human genetic diseases. Among these they mentioned Huntington disease as being a big genetic challenge. This surprised me because here in France this disease was not on the agenda of medical geneticists.

PSH. Yes. Maybe this reflects the paediatric origins of medical genetics in France.

JCK. Yes. You are right. This point is important if you make a comparison between our countries..

PSH. Yes. It's something which has come across very strongly, and I was told also that Robert in Lyon had great difficulty in being accepted, because he was an adult physician and neurologist.

JCK. Yes I knew Jacques-Michel Robert. He was quite interested in DMD. But he was completely rejected by Frézal.

PSH. So you felt this gave you the opportunity now to come back to muscle disease with new tools.

JCK. Yes. It seemed to me that an unsolvable enigma such as DMD would be the first to benefit from the gene approach. Putting one's finger on the DMD gene did no longer seem utopic.

PSH: Coming back to AFM and your links there.

JCK. As I told you, when the DMD locus was mapped by linkage analysis I approached the AFM, trying to convince its President, Bernard Barataud that it was a real breakthrough, and that further progress depended strongly on the cooperation of patient's families. I remember our first meeting. He was in my office with two other co-founders of AFM (René Cadoret and Michel Pignolet) and I explained them on a blackboard the principle of linkage analysis and

chromosome walking. When I finished Bernard stared at me and said: "*I did not understand a single word of your explanations, but I have the feeling that we have to trust this strategy that may eventually bring us to the gene"*. Then he added that this was in contrast with the junk he had heard for so many years and from so many charlatans.

PSH. This is the value of someone directing these foundations who has the ability to see what is needed.

JCK. Yes, but Bernard Barataud was exceptionally clear sighted. Did you ever meet him?

PSH. Not for interview here, no.

JCK. For your project you should.

PSH. It's like I said, every person I have talked with gives me the names of 5 more people, and you are no exception.

JCK. You know he is the man who built the first industrial laboratory facility called Généthon. This entirely novel organisation produced in an astounding short period of time the first human genome linkage and physical maps, with the help of the CEPH (founded by Jean Dausset), and two big brains: Jean Weissenbach and Daniel Cohen.

PSH. Absolutely, and I think this to me is extraordinary, not just for muscle disease but how he reunited it with the mainstream of the building of a general morbid map of the genome.

JCK. I can give you another trait of his amazing ability to anticipate. Two or three years after having launched the Généthon and harvested the first fruits, he convened the scientific council of the AFM chaired by François Gros, and urged us to do some brainstorming to imagine the future of this enterprise. Before having finished with Généthon I he was already dreaming of Généthon II!

PSH. Yes, now that's a true genius. Jean-Claude, we must finish soon but there are a couple of things I would like to ask. I have spoken with a number of people about their early experiences in France in relation to World War II, because for you, especially for someone with a Jewish background, these must have been years of devastating problems and am I right this must have had major influences on you. Is it something which you feel comfortable to say anything about?

JCK. Well, it is too long a story to be told here. Let me just say that I was almost 10 years old in June 1940 when the disastrous defeat occurred. It

resulted in the fall of the legal Republic regime and the installation of a proGerman government headed by Maréchal Pétain, settled in Vichy and ruling the Southern half of France, not occupied by Germans. We had fled from Paris to Nice where we stayed until the Germans came in (September 1943). Until that date we could lead a normal life, but then we had to hide with false papers. We left Nice to Montpellier and then to Millau, a small town in Aveyron, where we were hidden and helped by a network of people, who were mostly Protestants. They obeyed their moral principles and did not pretend to be heroes. But what they did was heroic, because they exposed their life. Thanks to them we escaped deportation. Other members of the family in Paris and Marseille were not as lucky. Six of them were deported and only one survived. However 75 % of the Jewish community in France escaped extermination, a proportion remarkably high. This is explained by the general reluctance of the non-Jewish population to cooperate with the anti-Semitic policy. Among them about 3,000 have been officially distinguished and received the Israeli medal of “Righteous among the Nations”, but the great majority of French people who saved Jews acted individually at their own personal level. They are the unknown “righteous” soldiers.

PSH. Jean-Claude, thank you so much and I think I will finish recording and thank you for sharing so much with me. It really is a privilege and something which is quite different from what I have heard from other people.

JCK. Listen Peter, I really appreciate the way you managed this interview. Thanks for your patience

PSH. Thank you Jean-Claude.

End of interview

[This edited transcript contains extensive changes from the original interview, which is also preserved in the project archive]