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Prof. Christine Petit is Bringing the Deaf out of the World of Silence

Interview published in Le Point on 1 April 2014

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EDITOR'S NOTE

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- Christine Petit's work on hearing and hereditary deafness, of major importance for hearing-impaired young people, should also improve the life of the elderly. The cochlea, the sensory organ of hearing, is virtually an open book for Christine Petit, professor at the Collège de France and the Institut Pasteur, and head of the INSERM Unit 1120 (Genetics and Physiology of Hearing). She is behind the discovery of a great number of genes responsible for deafness and associated faulty mechanisms, as well as the deciphering of this organ's functioning at molecular level.**

What was the starting point for your work?

Christine Petit I was always interested in sensory perception and communication between individuals. After studying olfaction, I decided to focus on audition. At the time, we had knowledge on the functioning of the hearing system, particularly that of the cochlea, but no data to understand the molecular mechanisms of its development or functioning. This was due to the very small number of cells for each of the cellular types of which it is comprised. Upon reflection, it became clear that only the genetic approach, in other words searching for the genes whose deficiency causes deafness, could allow us to understand its functioning at cellular and molecular level. Unlike all the other approaches, its effectiveness was not contingent on the number of cells and molecules involved in the process we sought to explain.

How did you progress in your area of expertise and with what results?

C. P. My team and I first resolved the different problems that prevented us from identifying the genes responsible for deafness in humans, by analysing very large families suffering from deafness, living in remote areas.

We now know barely one hundred genes involved in isolated deafness and about two hundred for deafness associated with other clinical signs (syndromic deafness). We thus broke down deafness into all these different forms. The next stage was to understand how, for each of them, the defective gene affects hearing. This could only be achieved using animal models of these forms of deafness: mice in which the same gene as the one responsible for deafness in humans is deactivated. Today, the different forms of deafness can be categorized into a few main pathogenic groups. In parallel, these data shed light on the cochlea's normal functioning.

We are now able to diagnose the genes responsible for deafness in a child or young adult, which allows us to ascertain the hereditary nature of this problem and thus to inform families of the existence of a risk for future children. The advances made through the study of animal models of deafness have led to changes in the interpretation of certain audiometric tests commonly used. For each form of deafness whose gene has been identified, these models also make it possible to identify whether or not the use of hearing aids (conventional prostheses or cochlear implants) will be beneficial.

What will you need in the future for research in your field to progress?

C. P. With regard to hearing impairments, we have yet to discover the genes of susceptibility to age-related sensorineural hearing loss, presbycusis, affecting 10 to 15% of the general population. As in the case of premature deafness, this is a prerequisite for us to understand the mechanisms and thus develop prevention and treatment tools tailored to each form. There is probably a large number of such forms. Research must also develop the currently fledgling knowledge of the processing of acoustic signals at brain level and of auditory perception. This is crucial for understanding tinnitus and hyperacusis. Finally, the time has come to develop therapeutic alternatives to hearing aids. While therapeutic advances are now a realistic objective, this hope must be associated with preventive measures against auditory stressors, primarily overexposure to noise.

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Genetics and Cellular Physiology