Sarah Vaughn Joins Pops For Jazz

"Sassy" Sarah Vaughan joins Arther Fiedler and the Boston Pops for an evening of classical music, popular music and - naturally, with the "divine Sarah" - jazz. It's 'Evening at Pops '76' Sunday, Aug. 29 at 8 p.m. and repeating Tuesday, Aug. 31 at 9 p.m. on TV-10.

The Pops Orchastra plays two Richard Wagner compositions for openers, followed by "Hora Staccato." When Sarah Vaughan enters, the lights dim, the stage darkens and she captures the audience with the beautiful song "Wave." Then, with just her trio for backup, Sarah swings into "Day In, Day Out," followed by a slow, poignant rendition of "Rainy Days and Mondays." It never sounded quite so sad, quite so mellow, quite so good.

"The summer knows/the summer's wise/she sees the dust winthin our eyes." sings Sarah. With the full Pops Orchestra behind her, the blues number "The Summer Knows" is like a new song. Then, changing the mood once more, she performs "I'll Remember April," complete with scatting. She also sings, "I've Got a Crush on You," slow, but playfully letting her voice experiment with the arrangement, and "A Foggy Day in London Town." The sun was shining... everywhere!

Maestro Fiedler and the orchestra top off the show with "Mah-Na, Mah-Na" and the saucy hit "Mame."

Birth Defects Research Holds Key to Many Adult Diseases

by Samuel J. Ajl, Ph.D.
Vice President for Research
The National Foundation-March of Dimes

How many people picture a grown man or woman when we talk about birth defects?

Very few. A deformed infant or crippled child is the usual image.

Yet many birth defects, unsuspected in childhood and youth, surface later in life as heart failure, stroke, diabetes, and emphysema. March of Dimes-supported research into causes and treatment of birth defects includes what may be some of the most promising attacks on adult diseases.

For example, about one in 500 Americans—440,000 people—has an inherited tendency to very high blood cholesterol. The long scientific name, familial hypercholesterolemia, can be abbreviated simply to FH. This abnormality, strongly associated with clogging of blood vessels supplying the heart and brain, often leads to death from heart attack or stroke.

Cellular Defect

March of Dimes grantee, Dr. Joseph Goldstein, assistant professor of internal medicine at the University of Texas Southwestern Medical School, Dallas, is working on ways to suppress cholesterol production by cells of the liver and other organs. He has learned that FH results from inherited lack of "receptors" on the surface of these cells. The receptors' job is to carry blood cholesterol into the cell and order temporary halts in cholesterol production.

Dr. Goldstein is experimenting with compounds chemically similar to cholesterol which bypass the defective receptor mechanism and suppress cholesterol production in human cells grown in the test tube. Discovery of a compound which is effective for long periods, without harmful side-effects, may prove useful in treating not only FH, but other high-cholesterol conditions as well.

Diabetes may result from a number of separate or interacting factors, but heredity is clearly among them. Although most diabetics respond well to insulin, many suffer slow degenerative disease of the eyes, kidneys, heart, and blood vessels. This results in chronic illness, pain, kidney failure, blindness, and early death.

New Drug

In Boston, MOD grantee Dr. Kenneth Gabbay, associate in endocrinology at Children's Hospital Medical Center, suspects that these symptoms are caused by accumulations of sugar-alcohols which may be by-products of diabetes.

He is studying a new drug which prevents such tissue damage in animals, apparently by blocking an enzyme which produces sugar-alcohols. The experimental drug appears safe for animals and may prove effective in sparing diabetic humans from long-term degenerative effects of their disorder.

UNKNOWN TO THEM, three seemingly healthy adults in this group are destined to suffer late-onset birth defects. More and more, research shows that inherited factors contribute to "adult" diseases such as stroke, diabetes, heart disease, and emphysema.

Third Target

Emphysema is a common, progressive disease in which loss of elasticity in the lungs' air sacs causes increasingly difficult breathing and consequent strain on the heart. It is a frequent cause or contributing factor in disability and death among adults.

Recent findings indicate that a major cause of emphysema is an inherited deficiency of the substance known as alpha-1-antitrypsin (AAT).

The natural function of AAT is to inactivate trypsin and other protein-destroying enzymes. Trypsin released by white blood cells, possibly to attack bacteria, may also attack lung tissue unless prevented by AAT.

Research in AAT-deficient emphysema is still at a basic stage. Another NF grantee, Dr. Irving Crawford, microbiologist at Scripps Clinic and Research Foundation in La Jolla, California, is studying inherited variants of the molecular structure of AAT which may determine severity of emphysema.

Clarification of AAT variants could make it possible to identify individuals at special risk early in life. They could then be protected against seriously aggravating factors such as respiratory infections, and warned about smoking and living or working in heavily air-polluted areas.

This investigation may provide important clues to origins, prevention, and treatment of AAT-deficient emphysema.

Partly because of the time lag between genetic cause and late onset, many inborn conditions which contribute to adult disease are not yet well understood, and probably many others remain to be discovered. As we learn more about these, the benefits of birth defects research will extend to more and more people, including many seemingly healthy individuals destined to be affected by birth defects long after childhood.