WEBVTT

00:00:04.504 --> 00:00:06.589 Lipoproteins are small particles

00:00:06.589 --> 00:00:09.843 that are needed to transport lipids across the body

00:00:09.843 --> 00:00:12.178 and deliver these lipids to all the organs.

00:00:12.637 --> 00:00:16.141 But when these lipids are accumulated in blood stream in high levels,

00:00:16.141 --> 00:00:21.563 particularly the low density lipoproteins that carry a very large amount of cholesterol,

00:00:21.855 --> 00:00:25.567 they can be accumulated in wrong places like the arterial wall,

00:00:25.942 --> 00:00:30.488 and cause the initiation of formation of arthroscopic plaques.

00:00:31.072 --> 00:00:36.911 We also carry lipids in another good lipoproteins called high density lipoproteins.

00:00:37.078 --> 00:00:41.249 They have an opposite role and promote the uptake in lipids

00:00:41.249 --> 00:00:44.335 in the periphery, and retrieve these lipids back to the liver.

00:00:44.377 --> 00:00:49.049 Then this is the thing that we know is a good cholesterol that is thought

00:00:49.049 --> 00:00:52.677 to play an important role in protecting cardiovascular disease.

00:00:55.764 --> 00:00:58.516 My lab studies atherosclerosis

00:00:58.516 --> 00:01:02.312 which is a very complex disease that involved alteration in metabolism,

00:01:02.312 --> 00:01:05.315 being the accumulation of this bad cholesterol,

00:01:05.315 --> 00:01:07.067 LDL cholesterol in the arteries,

00:01:07.067 --> 00:01:10.779 the major factor that initiated the formation of these lesions.

00:01:10.987 --> 00:01:13.865 Then my lab is working in identifying novel

00:01:13.865 --> 00:01:17.160 molecular mechanisms that control lipoprotein metabolism,

00:01:17.160 --> 00:01:21.122 and in this regard, we discover a number of novel molecules

00:01:21.122 --> 00:01:24.459 called micro RNAs, playing an important role here.

00:01:24.459 --> 00:01:28.004 And also another set of molecules called angiopoietin lipoproteins.

00:01:28.004 --> 00:01:32.717 that control catabolism or recycling, of these lipoproteins in circulation.

00:01:33.093 --> 00:01:36.679 In top of that, we also are very interested in studying

00:01:36.679 --> 00:01:41.518 the chronic inflammatory response that occurs in the vessels where these lesions develop.

00:01:42.102 --> 00:01:44.854 Particularly interested in the role of these lipids

00:01:44.854 --> 00:01:49.359 modifying the innate immune responses that are important

00:01:49.359 --> 00:01:54.322 for the progression and the rupture of these lesions in the large arteries.

00:01:57.700 --> 00:02:01.412 We use models of living organisms that develop this disease,

00:02:01.412 --> 00:02:05.959 where we can study the initiation and progression of this pathology.

00:02:06.459 --> 00:02:09.796 We combine this with very basic vascular biology,

00:02:10.046 --> 00:02:13.842 cell biology, transcriptomics, single cell RNA sequencing,

00:02:13.883 --> 00:02:17.762 lipoprotein kinetics, lipoprotein fractionation, lipidomics,

00:02:17.762 --> 00:02:22.642 and also we collaborate with physican-sciencists to study

00:02:22.642 --> 00:02:25.770 the pathology also in human tissue as well.

00:02:28.815 --> 00:02:30.775 We know for the last two decades,

00:02:30.775 --> 00:02:34.362 that the statins has been very effective to protect against cardiovascular disease

00:02:34.362 --> 00:02:39.159 by significantly reducing the levels of LDL cholesterol.

00:02:39.200 --> 00:02:41.786 But still, there is a residual risk,

00:02:41.786 --> 00:02:44.956 and this is why we need a very specific mechanism

00:02:44.956 --> 00:02:47.292 that has to be very tightly regulated,

00:02:47.292 --> 00:02:50.295 that allow us to transport lipids to all the organs.

00:02:50.295 --> 00:02:55.008 But at the same time, have another good mechanism to rid out of cholesterol

00:02:55.008 --> 00:02:59.554 when we don't need more, then this is where the potential discovery

00:02:59.554 --> 00:03:05.768 of novel therapies that can protect attenuate the progression of disease.