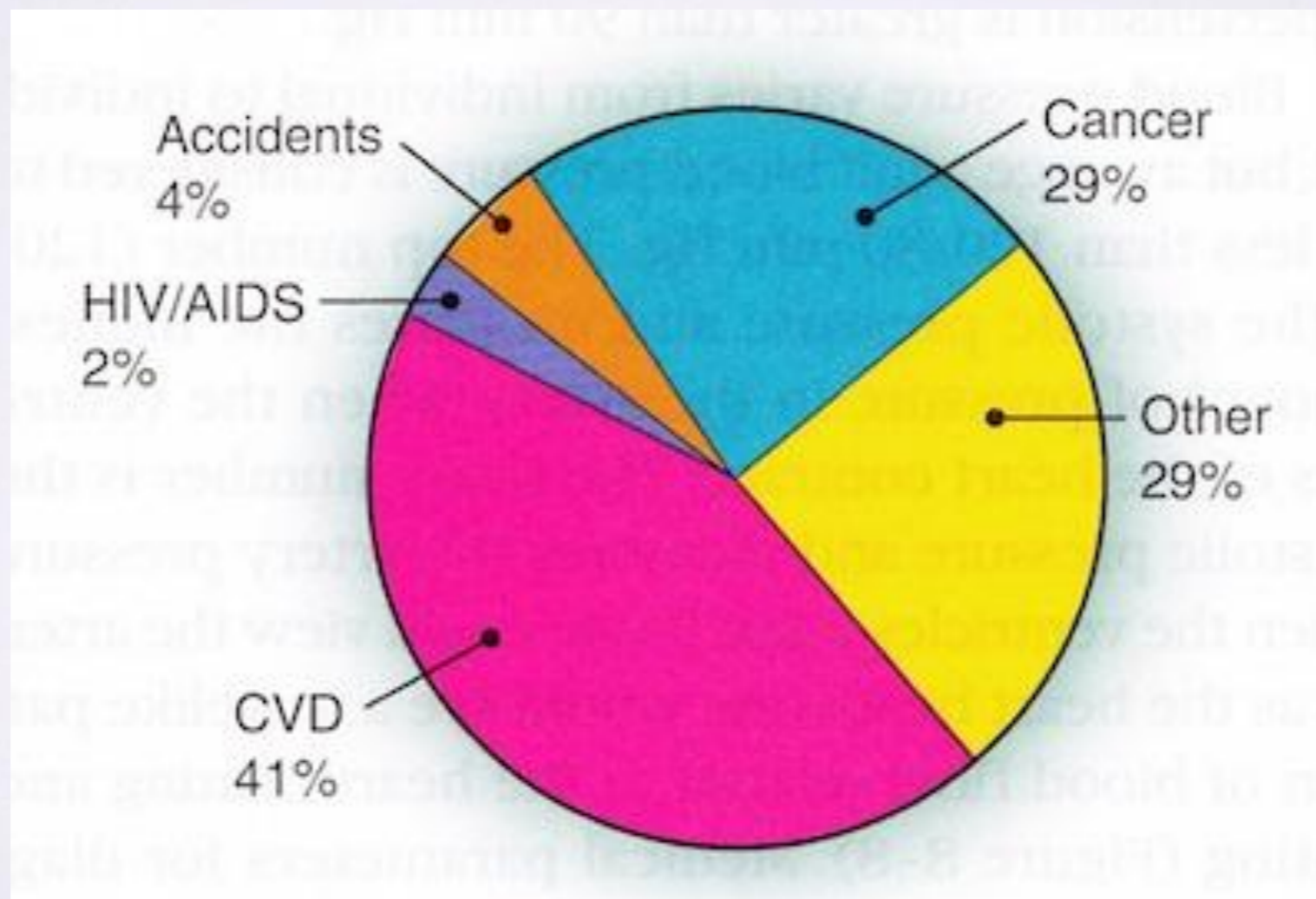


# CLASSIFICATION AND THE SURVIVAL ANALYSES IN THE ARTREAT PROJECT

Goran Rakocevic  
Zoran Babovic  
Marko Novakovic  
Nenad Korolija  
Veljko Milutinovic

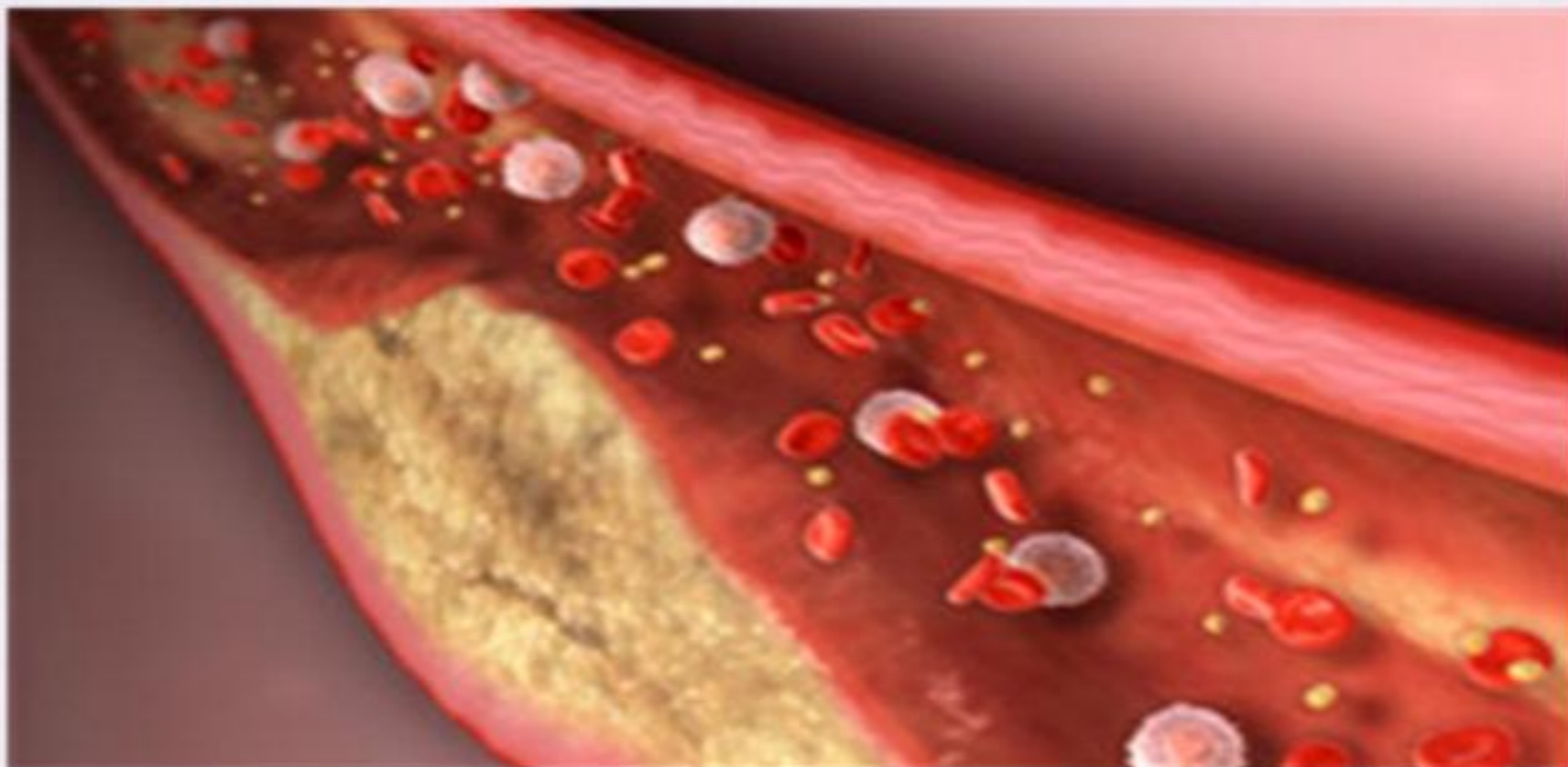
# ATHEROSCLEROSIS

- Why is atherosclerosis important?
- It is a CVD (Cardiovascular Disease)



# ATHEROSCLEROSIS

- Atherosclerosis is the condition in which an artery wall thickens as the result of a build-up of fatty materials such as cholesterol

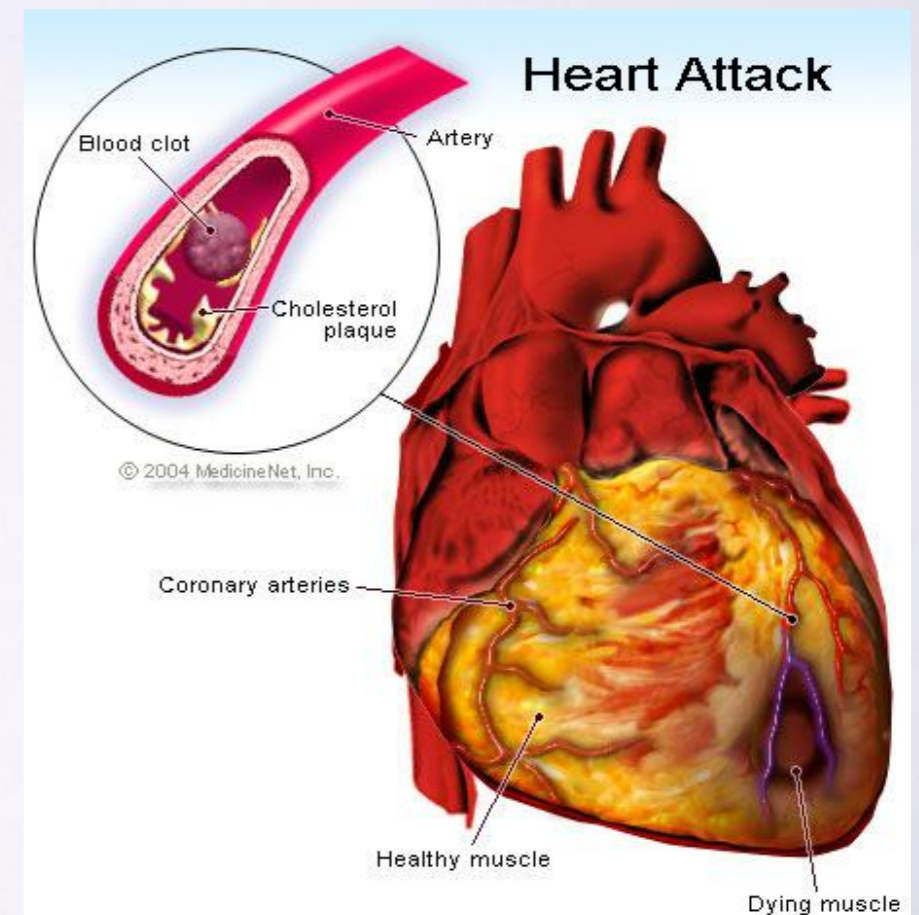


# ATHEROSCLEROSIS

- Etiology: Injury to the artery wall initially patched by LDL plasma proteins
  - LDL plasma proteins invite white blood cells which cause an inflammatory response and finalize the patching of injured arterial walls
  - Cholesterol and triglycerides attached to the LDL proteins are not removed by HDL
  - The cholesterol oxidizes and hardens, stiffening the arterial wall,
    - Irreversible hardening of arteries

# ATHEROSCLEROSIS

- Atherosclerosis is chronic and cumulative
- How atherosclerosis affects the organism?
  - obstruction of blood flow causing slow tissue death.
  - plaque ruptures forming thrombus
    - complete blockage?
    - tissue death within 5 minutes.



\*The tissue involved is that which is fed by the blocked artery. \*This thrombus related tissue death is what we call an infarction. This same process within the blood vessels of the brain is called a stroke.

# ATHEROSCLEROSIS

- Why is atherosclerosis interesting for research?
  - **affects the entire artery tree,**
    - the coronary
    - Renal
    - Femoral
    - Cerebral
    - Carotid arteries.

# THE ARTREAT PROJECT

- ARTreat targets at providing a computational model of the cardiovascular system, to improve prediction for the atherosclerosis progression and propagation into life-threatening events.
- An FP7 Large-scale Integrating Project (IP)
- 16 partners
- Funding: 10,000,000 €

# ARTREAT GOALS

- ARTreat provides a **three-level patient model** describing the 3d arterial tree anatomy, the patient-specific blood flow and blood particle dynamics and the biological processes that lead to the creation and progression of atherosclerotic plaques.
- Provide patient specific model of arterial tree for clinical decision support and training
- Etiology of Atherosclerosis (e.g. genetic, fatty intake etc.)





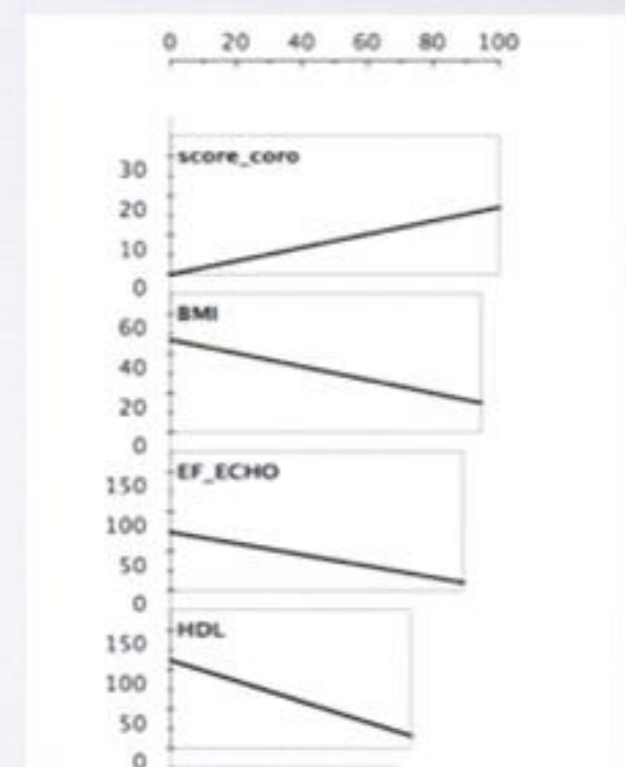
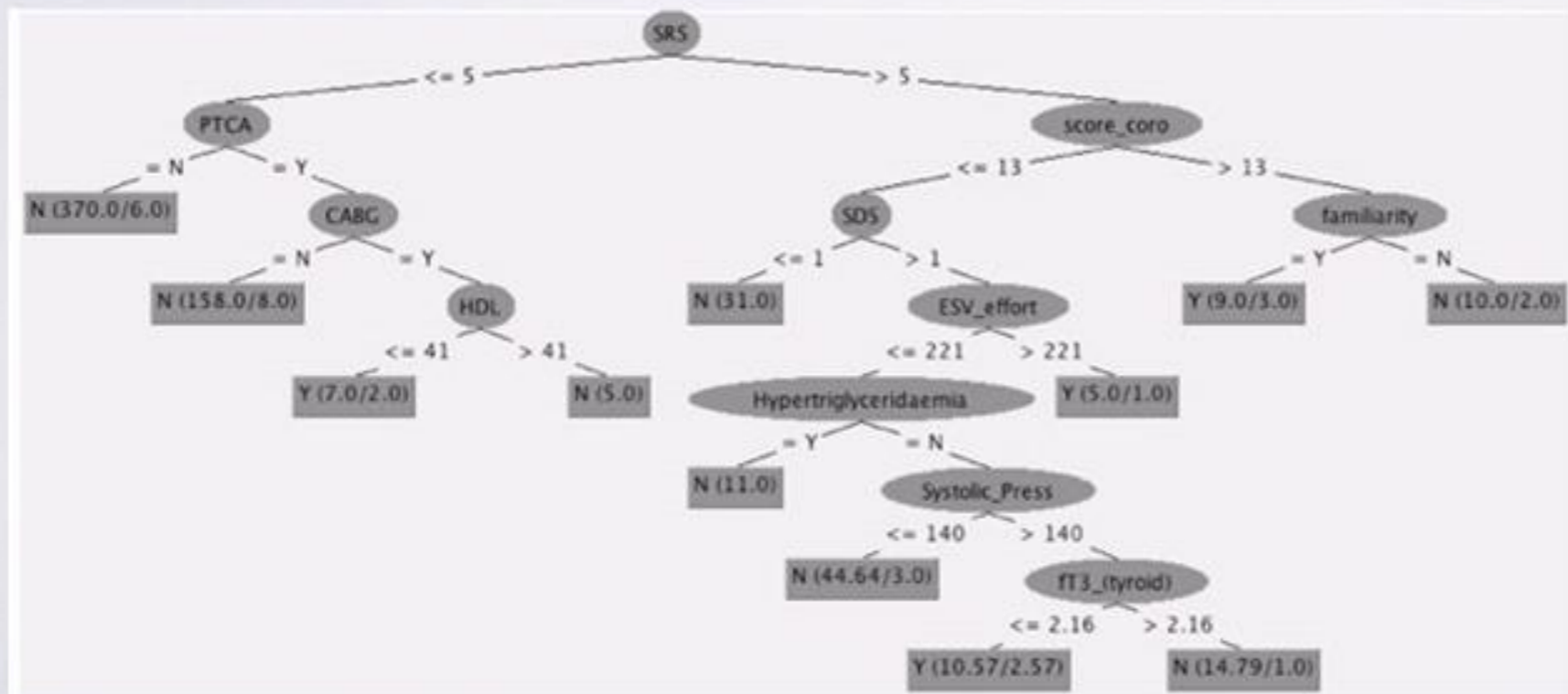
# THE DATA

- 3000 patients with 97 attributes
- 450 patients with genetic profiles
- 400 patients with repeated angiographies
- 600 patients with scintigraphies

V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ	AK	AL	AM	AN	AO	AP	AQ	AR	AS	AT	AU	AV	AW	AX				
11/18/82	2	75																														
1/18/83	3	75							75																60	80	120	1.1	264			
12/11/78	2	90											50												64	80	115	1.0	230			
2/4/81	1	100	100																						60	70	110	1.2	179			
3/14/83	1	90	90																						95	80	110	0.6	144			
5/9/79	2	90						90																	64	82	142	1.0	220			
11/3/83	3	100							100	100															70	70	110	1.59	139	33		
2/14/83	1												90	75	90											65	70	104	1.4	270		
2/14/83	0																									66	85	120	1.0	230		
4/22/83	2	90																							70	75	115	1.1	223			
3/2/83	3	90	90						75				75	50	50										60	100	170	1.4	248			
3/6/83	4	50			50								75	100	90	100									50	48	90	1.08	236	53		
3/30/83	3	90							90				90	90		90									75	84	90	1.5	238			
4/20/83	2	100						90	90	90															74	80	130	1.7	267			
5/15/79	3	100											100													68	79	118	0.8	415		
11/14/87	3	90			90		50	75	100				100													64	80	140	1.49	142	29	
4/27/83	3	90	90																							62	82	140	1.1	320		
3/9/83	4																									100	70	80	1.1	215		
5/4/83	1	90																								64	80	120	0.9	225		
5/13/83	0																									58	80	118	1.2	251		
6/8/83	1																									60	85	130	1.0	171		
7/13/83	0																									62	85	125	1.2	223		
7/15/83	2	100							90																	54	80	120	1.1	189		
5/11/83	3	100																								58	80	140	1.3	209		
4/22/88	4	100								100																100	64	70	135	1.1	140	45
10/5/83	2	100																								62	70	110	1.0	294		
10/28/83	2	90																								78	85	122	0.9	239		
11/11/83	1									75																76	80	120	0.8	250		
2/8/84	3	75		75					100	100																90	90	135	1.0	195		
12/14/83	3	50																								76	76	100	1.0	271		
11/2/83	1																									50	50	100				
11/14/83	4	90	75	75	90				90																	75	70	86	1.0	235		
1/23/84	1																										72	80	120	0.9	218	
10/9/82	1	100		100																							80	80	140	0.92	149	44
3/12/84	2	50	50					75	50					90	100												58	80	120	1.2	224	
3/23/84	0																										72	88	138	0.8	307	
2/27/84	1									75	100			100	90												64	70	110	1.1	257	
3/7/84	1	50			50	90	90	90																			76	75	124	1.3	189	
3/28/84	3	100																									78	78	118	1.0	208	
3/28/84	4	90		90																							50	80	90	1.4	217	

# CLASSIFICATION: HARD EVENTS

- Divide patients into:  
those who had an event vs. those who did not  
(group patients with similar anamnesis together)



# CLASSIFICATION: HARD EVENTS

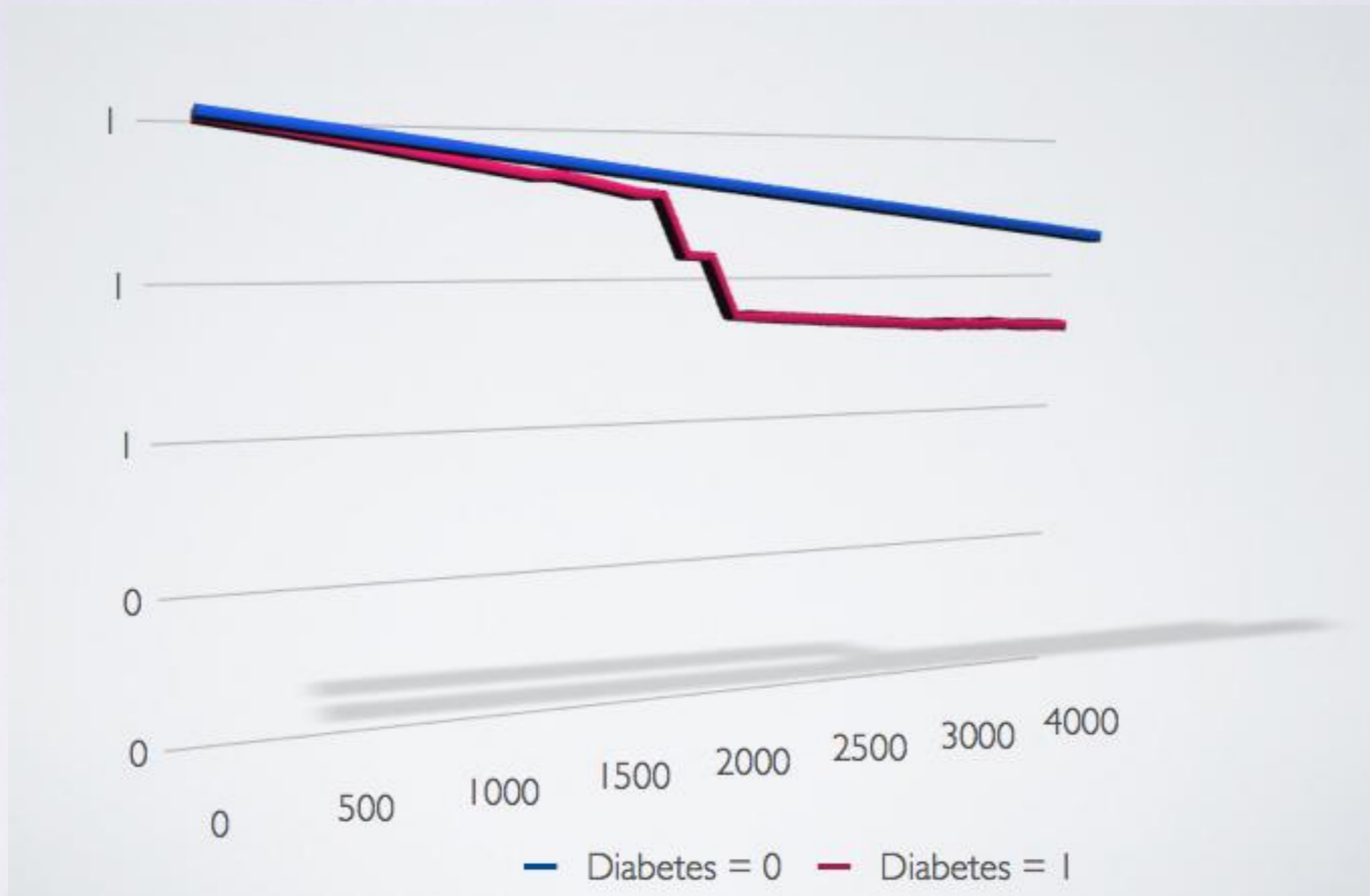
- Interventions in most severe cases change patient characteristics (they change the anamnesis)
- Hard event classification became meaningless
- A \*MACE study (Major Adverse Cardiac Events)
- Better results, still not sufficient

\*objective measures of acute and/or adverse cardiovascular events which are used to assess the effects of various interventions (e.g., rotablation, angioplasty, stenting) or therapeutics on outcomes, in the context of a clinical trial.

# SURVIVAL ANALYSES

- Type of problems:
  - Study over a period of time
  - In the course of study an event either happens or not
  - Not just about if, but also when

# THE KAPLAN-MEIER ESTIMATOR



an estimator for estimating the survival function from lifetime data.

# COX REGRESSION

- Proportional Hazards assumption
- Omits the underlying hazard function
- Gives only a Hazard Ratio
- Relatively Simple, and often sufficient
- Easy to add time varying predictors and covarities

**END**

**Thank You**