Supplementary Material

1 Supplementary Table 1

Table 1. Criteria in men.

ID	VARIABLE	UNITS	CLINICAL CRITERIA		PROJECT 42*		CLINICAL CRITERIA REFERENCE
			low	High	min	Max	
D0	sex: man				1	1	
D1	age	years old			18	25	
P0	systolic blood pressure (SBP)	mmHg	90	120	90	120	(Whelton et al., 2017)
P1	diastolic blood pressure (DBP)	mmHg	60	80	60	80	(Whelton et al., 2017)
DP0	pulse pressure (PP)	mmHg	20	60	30	60	(Whelton et al., 2017)
DP1	mean arterial pressure (MAP)	mmHg	70	93	70	93	(Whelton et al., 2017)
T0	axillary temperature	°C	35.5	37.0	35.5	37.0	(Sund-Levander et al., 2002)
O2	oxygen saturation	%	90	100			(O'Driscoll et al., 2017)
O3	PAO_2FIO_2 arterial oxygen pressure/inspired fraction of oxygen	mmHg/%	350	450			(Hübler et al., 2014)
BR	breathing rate	bpm	8	30			(Metlay et al., 2019)
HR	heart rate	bpm	50	100			(Kusumoto Fred M. et al., 2019)

B0	weight	kg			46.1	78.5	
B1	height	m	1.60		1.60	1.85	(Grimberg et al., 2016)
DB1	body mass index (BMI)	kg/m^2	18	25	18	25	(Almeda-Valdes et al., 2016)
M1	triglycerides	mg/dL	40	150	40	126	(Baigent et al., 2019)
M2	total cholesterol	mg/dL		200	109	200	(Mach et al., 2020)
M3	HDL cholesterol	mg/dL	40	90	44.6	67.3	(Mach et al., 2020)
M4	LDL cholesterol	mg/dL		116	58	109	(Mach et al., 2020)
M5	glucose	mg/dL	70	100	70	94	(Alberti K.G.M.M. et al., 2009)
M8	blood urea nitrogen (BUN)	mg/dL	9	23	9	23	(Tyagi & Aeddula, 2021)
M10	serum creatinine (SCr)	mg/dL	0.8	1.5	0.8	1.2	(Hosten, 1990)
M12	HbA1c	%		5.7	4.7	5.7	(American Diabetes Association, 2020)
M13	C-reactive protein (PCR)	mg/dL		1	0.009	0.323	(Oda et al., 2006)
M20	direct bilirubin	mg/dL	0	0.3	0.1	0.3	(G.-M. Zhang & Hu, 2018)
M21	indirect bilirubin	mg/dL	0.09	0.65	0.09	0.65	(G.-M. Zhang & Hu, 2018)
M22	aspartate aminotransferase (AST)	UI/L	5	35	18	28	(Lala Anuradha et al., 2020)

M23	alanine transaminase (ALT)	mU/mL	5	35			(Lala et al., 2021)
M24	Albumin	g/dL	3.5	5.0			(Kopple et al., 2012)
M25	lactic dehydrogenase	U/L	45	90			(Koukourakis et al., 2009)
M26	creatinine kinase	U/L	20	215			(Laoutidis & Kioulos, 2014)
M27	creatinine kinase MB	ng/mL	5	25			(Kurapati & Soos, 2021)
M28	procalcitonin	ng/mL		0.05			(Cleland & Eranki, 2021)
M29	ultrasensitive troponin	pg/mL		36			(X. Zhang et al., 2019)
M30	natriuretic peptides (BNP)	pg/mL		100			(Potter et al., 2009)
M31	interleukin-6 (IL-6)	pg/mL	5	15			(Alecu et al., 1998)
M32	thyroid stimulating hormone (TSH)	mU/mL	0.4	4.8			(Khandelwal & Tandon, 2012)
M33	vitamin D	ng/mL	30	100			(Sizar et al., 2021)
M34	ferritin	ng/mL	20	200			(Wang et al., 2010)
M35	dimer D	ng/mL		500			(Bounds & Kok, 2021)
M36	prothrombin	s	10	12.5			(Lala et al., 2021)
M37	fibrinogen	mg/dL	150	400			(Roshal, 2013)
DM1	estimated glomerular filtration rate (eGFR)	ml/min	90	120	90	120	(Musso et al., 2016)

DM2	estimated average glucose (eAG)	mg/dL			88	120	(Guo et al., 2020)
DM3	eAG-fasting glucose	mg/dL			8	32	(Guo et al., 2020)
DM4	BUN to creatinine ratio		8.17		8.18	21.36	(Haines et al., 2019)
DM5	METS-IR			51.13	26.80	30.85	(Bello-Chavolla et al., 2018)
DM6	TyG index			4.65	4.05	4.65	(Simental-Mendía et al., 2008)
DM7	TyG-BMI index			135.4	86.2	115.7	(Lee et al., 2021)
DM8	triglycerides-HDL ratio			2.75	1.08	1.72	(Cordero et al., 2008)
H0	leukocytes	10^9/L	3.8	10.4	4.4	8.5	(Adeli et al., 2015)
H1	total neutrophils	10^9/L	1.9	8.0	2.0	5.9	(Coates, 2021)
DH1	total neutrophils percentage	%	40	70	40	70	(Coates, 2021)
H3	lymphocytes	10^9/L	1.5	3	1.5	3	(Rosenthal, 2020)
DH2	lymphocytes percentage	%	20	50	23	47	(Rosenthal, 2020)
DH6	neutrophils-lymphocytes relation		0.78	3.53	0.86	3.02	(Forget et al., 2017)
H8	hemoglobin	g/dL	12	18.7	14.8	17.9	(Adeli et al., 2015)
H14	platelets	10^3/µL	152	324	159	322	(Adeli et al., 2015)
H15	cytopenia	%	0				

CT1	average right atrial ventricular epicardial fat	mm			(Eroğlu, 2015)
CT2	average left atrial ventricular epicardial fat	mm			(Eroğlu, 2015)
CT3	average intraventricular groove epicardial fat	mm			(Eroğlu, 2015)
CT4	average epicardial fat	mm	1	9.5	(Iacobellis & Willens, 2009)
CT5	main trunk pulmonary artery diameter	mm		29	(Truong et al., 2012)
CT6	right main artery diameter	mm		19.8	(Bozlar et al., 2007)
CT7	left main artery diameter	mm		22.1	(Bozlar et al., 2007)
CT8	thoracic or extra-pericardial fat volume	cm^3			
CT9	thoracic subcutaneous adipose tissue	mm			
CT10	Hounsfield units	HU	0	20	
CT11	epicardial spill on tomography				
CT12	extrapericardial fat in tomography				
CT13	presence of steatosis on tomography				

CT14	type of tomography according to its characteristics 0=negative, 1=typical, 2=unspecific, 3=atypical
CT15	presence of ground glass opacity in tomography
CT16	consolidation in tomography
CT17	presence of grounded glass opacity and consolidation
CT18	presence of aerial bronchogram in tomography
CT19	presence of atelectasis in tomography
CT20	pulmonary distribution in tomography (0=central, 1=peripheral, 2=both)
CT21	lateral involvement in tomography (1=unilateral, 0=bilateral)
CT22	pulmonary zone involved in tomography

	(0=upper, 1=central, 2=lower, 3=generalized)		
CT23	degree of pulmonary involvement (0=no affection, 1=mild<20%, 2=moderate 20-50%, 3=severe≥50%)		
CT24	pulmonary thromboembolia		
CT25	evidence of PTE in tomography (1=lobar, 2=segmentary)		
COM	number of comorbidities	0	0
SYMN	number of symptoms include: fever, cough, dyspnea, myalgias, headache, anosmia, diarrhea, cardiovascular event.	0	0
SYMD	days with symptoms before arrival to hospital	0	0

* (Barajas-Martínez et al., 2021)

2 Supplementary Table 2

Table 2. Criteria in women.

ID	VARIABLE	UNITS	CLINICAL CRITERIA		PROJECT 42*		CLINICAL CRITERIA REFERENCE
			low	high	min	Max	
D0	sex: woman				0	0	
D1	age	years old			18	28	
P0	systolic blood pressure (SBP)	mmHg	90	120	90	120	(Whelton et al., 2017)
P1	diastolic blood pressure (DBP)	mmHg	60	80	60	80	(Whelton et al., 2017)
DP0	pulse pressure (PP)	mmHg	20	60	20	60	(Whelton et al., 2017)
DP1	mean arterial pressure (MAP)	mmHg	70	93	70	93	(Whelton et al., 2017)
T0	axillary temperature	°C	35.5	37.0	35.5	37.0	(Sund-Levander et al., 2002)
O2	oxygen saturation	%	90	100			(O'Driscoll et al., 2017)
O3	PAO_2FIO_2 arterial oxygen pressure/inspired fraction of oxygen	mmHg/%	350	450			(Hübler et al., 2014)
BR	breathing rate	bpm	8	30			(Metlay et al., 2019)
HR	heart rate	bpm	50	100			(Kusumoto Fred M. et al., 2019)
B0	weight	kg			43.7	73.2	
B1	height	m	1.50		1.50	1.73	(Grimberg et al., 2016)
DB1	body mass index (BMI)	kg/m^2	18	25	18	25	(Almeda-Valdes et al., 2016)
M1	triglycerides	mg/dL	40	150	44	150	(Baigent et al., 2019)
M2	total cholesterol	mg/dL		200	106	200	(Mach et al., 2020)
M3	HDL cholesterol	mg/dL	50	90	50	66.4	(Mach et al., 2020)
M4	LDL cholesterol	mg/dL		116	50	112	(Mach et al., 2020)
M5	glucose	mg/dL	70	100	70	90	(Alberti K.G.M.M. et al., 2009)

M8	blood urea nitrogen (BUN)	mg/dL	9	23	9	19	(Tyagi & Aeddula, 2021)
M10	serum creatinine (SCr)	mg/dL	0.5	1.1	0.55	0.94	(Hosten, 1990)
M12	HbA1c	%		5.7	4.6	5.6	(American Diabetes Association, 2020)
M13	C-reactive protein (PCR)	mg/dL		1	0.009	0.323	(Oda et al., 2006)
M20	direct bilirubin	mg/dL	0	0.3	0.1	0.3	(G.-M. Zhang & Hu, 2018)
M21	indirect bilirubin	mg/dL	0.09	0.65	0.09	0.65	(G.-M. Zhang & Hu, 2018)
M22	aspartate aminotransferase (AST)	UI/L	5	35	12	25	(Lala Anuradha et al., 2020)
M23	alanine transaminase (ALT)	mU/mL	5	35			(Lala et al., 2021)
M24	Albumin	g/dL	3.5	5.0			(Kopple et al., 2012)
M25	lactic dehydrogenase	U/L	45	90			(Koukourakis et al., 2009)
M26	creatinine kinase	U/L	20	160			(Laoutidis & Kioulos, 2014)
M27	creatinine kinase MB	ng/mL	5	25			(Kurapati & Soos, 2021)
M28	Procalcitonin	ng/mL		0.05			(Cleland & Eranki, 2021)
M29	ultrasensitive troponin	pg/mL		15			(X. Zhang et al., 2019)
M30	natriuretic peptides (BNP)	pg/mL		100			(Potter et al., 2009)
M31	interleukin-6 (IL-6)	pg/mL	5	15			(Alecu et al., 1998)
M32	thyroid stimulating hormone (TSH)	mU/mL	0.4	4.8			(Khandelwal & Tandon, 2012)
M33	vitamin D	ng/mL	30	100			(Sizar et al., 2021)
M34	ferritin	ng/mL	20	200			(Wang et al., 2010)
M35	dimer D	ng/mL		500			(Bounds & Kok, 2021)
M36	prothrombin	s	10	13			(Lala et al., 2021)

M37	fibrinogen	mg/dL	150	400			(Roshal, 2013)
DM1	estimated glomerular filtration rate (eGFR)	ml/min	90	120	90	120	(Musso et al., 2016)
DM2	estimated average glucose (eAG)	mg/dL			85	114	(Guo et al., 2020)
DM3	eAG-fasting glucose	mg/dL			5.9	37	(Guo et al., 2020)
DM4	BUN to creatinine ratio		8.17		10.34	31	(Haines et al., 2019)
DM5	METS-IR			51.13	25.61	33.91	(Bello-Chavolla et al., 2018)
DM6	TyG index			4.65	4.07	4.65	(Simental-Mendía et al., 2008)
DM7	TyG-BMI index			135.4	74.6	108.2	(Lee et al., 2021)
DM8	triglycerides-HDL ratio			2.75	0.79	2.47	(Cordero et al., 2008)
H0	leukocytes	10^9/L	3.8	10.4	4.6	10.4	(Adeli et al., 2015)
H1	total neutrophils	10^9/L	1.9	8.0	1.9	7.0	(Coates, 2021)
DH1	total neutrophils percentage	%	40	70	41	70	(Coates, 2021)
H3	lymphocytes	10^9/L	1.5	3	1.5	3	(Rosenthal, 2020)
DH2	lymphocytes percentage	%	20	50	20	49	(Rosenthal, 2020)
DH6	neutrophils-lymphocytes relation		0.78	3.53	0.88	3.53	(Forget et al., 2017)
H8	hemoglobin	g/dL	12	16	13	16	(Adeli et al., 2015)
H14	Platelets	10^3/µL	153	361	158	357	(Adeli et al., 2015)
H15	cytopenia	#	0				
CT1	average right atrial ventricular epicardial fat	mm					(Eroğlu, 2015)
CT2	average left atrial ventricular epicardial fat	mm					(Eroğlu, 2015)
CT3	average intraventricular	mm					(Eroğlu, 2015)

	groove epicardial fat				
CT4	average epicardial fat	mm	1	7.5	(Iacobellis & Willens, 2009)
CT5	main trunk pulmonary artery diameter	mm		27	(Truong et al., 2012)
CT6	right main artery diameter	mm		19.8	(Bozlar et al., 2007)
CT7	left main artery diameter	mm		22.1	(Bozlar et al., 2007)
CT8	thoracic or extra-pericardial fat volume	cm^3			
CT9	thoracic subcutaneous adipose tissue	mm			
CT10	Hounsfield units	HU	0	20	
CT11	epicardial spill on tomography				
CT12	extrapericardial fat in tomography				
CT13	presence of steatosis on tomography				
CT14	type of tomography according to its characteristics: 0=negative, 1=typical, 2=unspecific, 3=atypical				
CT15	presence of ground glass opacity in tomography				
CT16	consolidation in tomography				
CT17	presence of grounded glass opacity and consolidation				

CT18	presence of aerial bronchogram in tomography		
CT19	presence of atelectasis in tomography		
CT20	pulmonary distribution in tomography		
CT21	lateral involvement in tomography		
CT22	pulmonary zone involved in tomography (0=upper, 1=central, 2=lower, 3=generalized)		
CT23	degree of pulmonary involvement (0=no affection, 1=mild<20%, 2=moderate 20-50%, 3=severe≥50%)		
CT24	pulmonary thromboembolia		
CT25	evidence of PTE in tomography (1=lobar, 2=segmentary)		
COM	number of comorbidities	0	0
SYMN	number of symptoms include: fever, cough, dyspnea, myalgias, headache, anosmia, diarrhea, cardiovascular event.	0	0

SYMD	days with symptoms before arrival to hospital	0	0
VSO	supplemental oxygen therapy		
VS1	number of days with ventilatory support		

* (Barajas-Martínez et al., 2021)

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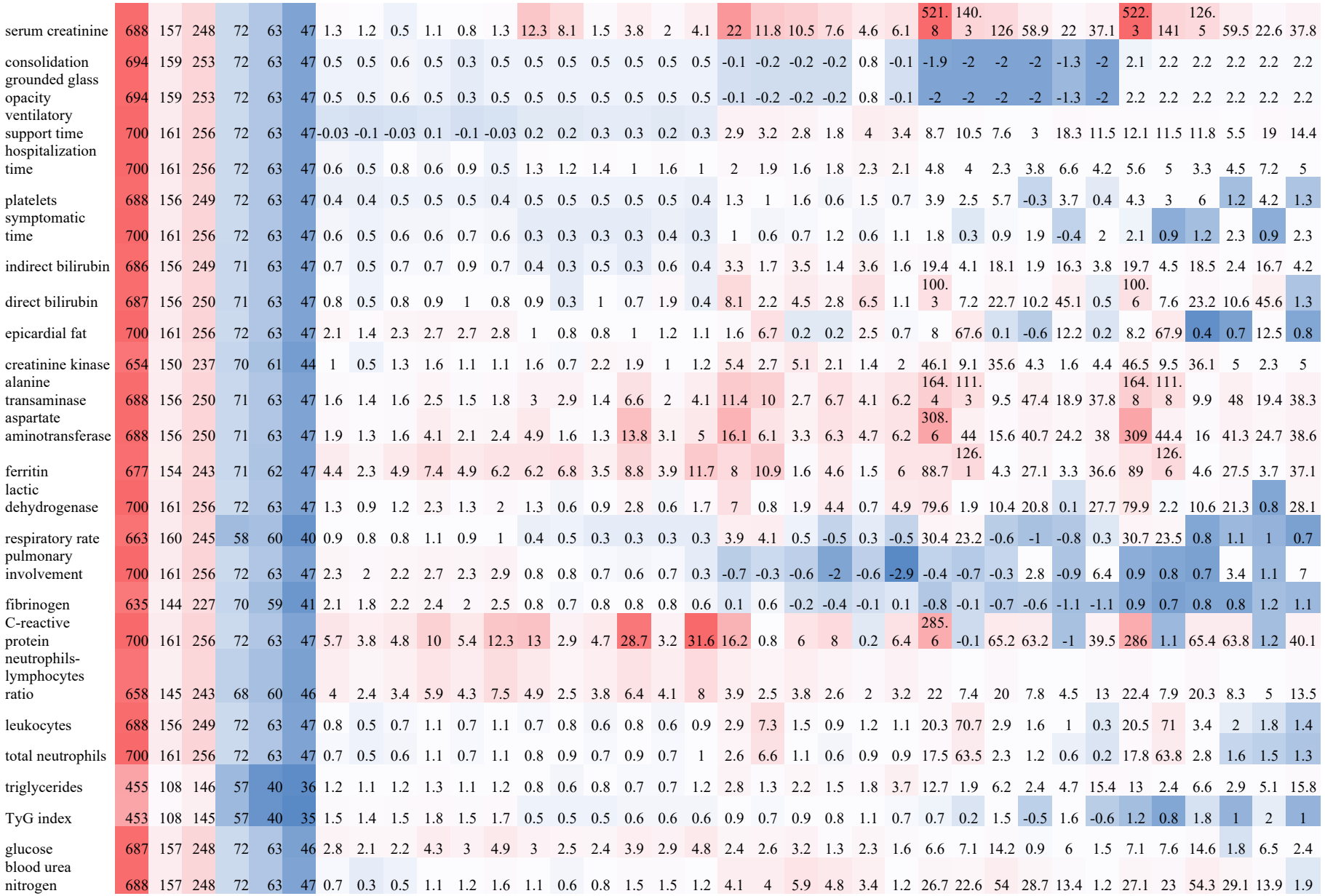
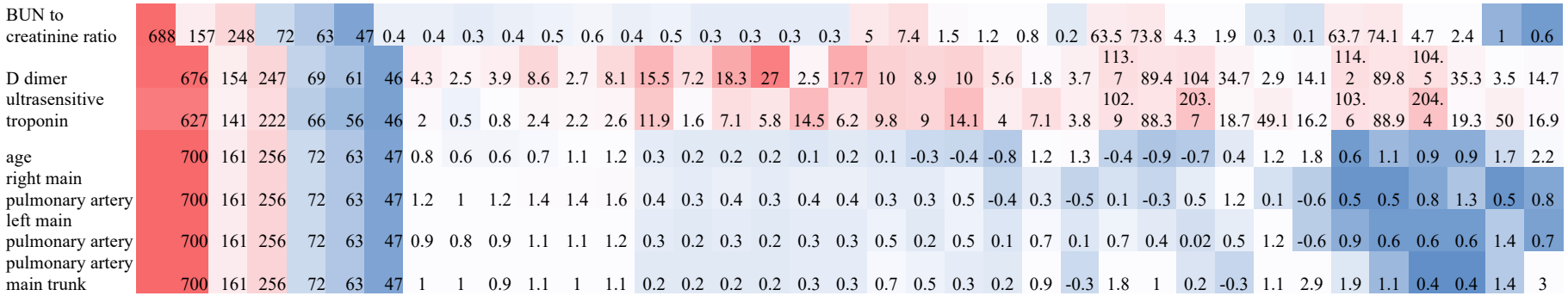
3 Supplementary Table 3

	Number of observations						Mean						Standard deviation						Skewness						Excess kurtosis						α					
	Full	FYR	MYR	MYD	MOR	MOD	Full	FYR	MYR	MYD	MOR	MOD	Full	FYR	MYR	MYD	MOR	MOD	Full	FYR	MYR	MYD	MOR	MOD	Full	FYR	MYR	MYD	MOR	MOD	Full	FYR	MYR	MYD	MOR	MOD
albumin	689	156	250	72	63	47	0.1	0.2	0.2	-0.03	0.1	-0.1	0.3	0.3	0.3	0.3	0.3	0.2	0.3	-0.1	-0.1	-0.1	0.7	-0.3	1.6	0.4	-0.3	0.1	0.2	-0.6	3	1.4	1.4	8.7	4.5	2.2
oxygen saturation	699	160	256	72	63	47	-0.9	-0.4	-0.5	-1.8	-0.7	-2.2	1.3	0.9	1	1.8	1.1	1.7	-1.6	-1.6	-2.3	-0.9	-2.4	-0.02	2.4	3.4	6.5	-0.1	7.3	-1.3	3.3	4.3	7.1	1.4	7.8	1.5
arterial pressure/inspired fraction of O_2	660	152	232	72	60	47	-1.3	-0.9	-1.2	-1.8	-1.3	-2	1	1	0.9	0.9	0.9	0.9	0.7	0.7	0.6	1.3	0.4	1	0.9	1.7	0.9	1.9	0.1	-0.1	1.4	2.2	1.3	2.3	0.8	1.1
est. glomerular filtration rate	688	157	248	72	63	47	-0.6	0.2	-0.6	-1	-1.1	-1.4	0.9	0.8	0.8	0.8	0.7	0.7	-0.3	-1.3	-0.2	0.2	-0.3	0.2	-0.4	3.4	-0.3	-0.5	-0.6	-0.8	1.7	5.6	1.4	1	1	1
lymphocytes	700	161	256	72	63	47	-0.5	-0.4	-0.5	-0.6	-0.6	-0.6	0.3	0.4	0.3	0.3	0.3	0.2	0.6	0.4	0.4	0.5	0.3	0.6	0.9	0.6	0.8	0.3	0.1	0.9	1.3	1.2	1.2	0.8	0.6	1.2
thoracic subcutaneous adipose tissue	700	161	256	72	63	47	0.3	0.4	0.2	0.3	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	1	0.7	1.4	1.9	1	0.5	1.5	1.5	2.8	6.7	0.5	-0.6	1.9	1.7	3.2	7	1.3	1
TyG-BMI index	426	103	130	56	39	35	1.3	1.3	1.2	1.5	1.1	1.2	0.5	0.5	0.4	0.6	0.4	0.4	1.4	1.4	0.7	1	0.9	0.9	4.3	4.9	0.8	1.4	0.8	0.6	4.5	5.1	1.1	1.8	1.2	1.1
body mass index	648	148	234	68	60	47	1.7	1.9	1.7	2	1.6	1.5	0.8	0.9	0.7	0.9	0.7	0.6	1.4	1.1	0.8	0.8	1.1	0.6	4.5	2.4	1.6	1.8	2.1	0.4	4.7	2.6	1.8	2	2.4	0.8
weight	656	148	237	70	60	47	1.1	1.1	1.2	1.4	1.1	1	0.5	0.6	0.5	0.6	0.4	0.3	1.2	1.3	1	1.1	0.3	0.6	3.5	3.4	2.6	2.1	-0.5	-0.5	3.8	3.7	2.8	2.4	0.7	0.9
hepatic steatosis	700	161	256	72	63	47	0.4	0.4	0.4	0.5	0.1	0.3	0.5	0.5	0.5	0.5	0.4	0.4	0.7	0.5	0.6	0	2	1.1	-1.5	-1.8	-1.4	-2	2	-0.8	2.1	2.2	2	2.3	3.8	2.2
heart rate	699	160	256	72	63	47	1	1	1.1	1.1	0.8	1.1	0.4	0.4	0.3	0.4	0.4	0.3	-0.9	-1.2	-0.05	-1.7	-0.7	-0.1	3.1	5.1	-0.3	4.8	1.2	0.02	3.2	5.2	0.4	5.1	1.5	0.3
axillary temperature	692	159	254	70	63	45	1.1	1.1	1.2	1.2	1	1.1	0.6	0.6	0.6	0.6	0.5	0.5	0.5	0.3	0.6	0.4	0.5	0.2	-0.1	-0.5	-0.4	-0.6	0.1	-0.6	0.8	0.8	0.8	0.9	0.7	0.8
prothrombin	662	150	238	70	60	45	0.8	0.5	0.8	0.9	1.6	1.8	2.5	0.4	0.5	1.7	5.5	6.5	15.1	1.3	0.8	7.3	6.4	6.1	247.8	3.4	0.5	55.3	42.8	37	248.3	3.7	1.1	55.8	43.4	37.7
vitamin D	522	113	193	54	49	35	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	0.1	0.1	0.1	0.1	0.1	0.1	1.5	1.1	1.6	0.01	0.7	0.5	5.2	1.7	6.2	-0.8	-0.01	-0.1	5.5	2.2	6.6	1.2	2.1	0.8
height	649	147	235	68	61	47	0.3	0.3	0.4	0.3	0.3	0.2	0.3	0.3	0.3	0.3	0.3	0.2	0.1	0.2	0.2	-0.5	-0.1	0.3	-0.2	-0.5	-0.2	0.2	-0.8	-0.2	1	1	0.8	1	1.3	1.3
hemoglobin	688	156	249	72	63	47	0.7	1.2	0.6	0.6	0.6	0.5	2.8	5.9	0.2	0.2	0.3	0.2	19.9	9.4	-1.1	-0.2	-0.9	-1.2	420.9	92.1	4	2	2.6	3.3	421.3	92.8	4.2	2	2.8	3.5
diastolic pressure	693	159	255	70	63	45	0.7	0.7	0.9	0.8	0.7	0.7	0.6	0.5	0.5	0.5	0.6	0.6	0.2	0.4	0.3	-0.1	0.04	0.6	0.2	0.4	0.3	-0.3	-0.2	0.2	0.8	0.9	0.7	0.7	0.8	1.2
mean arterial pressure	692	159	255	70	63	44	0.9	0.9	1	0.9	1	0.8	0.5	0.5	0.5	0.5	0.5	0.6	0.3	0.5	0.4	-0.1	0.3	1	0.4	0.5	0.5	-0.7	0.5	1.4	0.8	0.9	0.7	0.9	0.8	1.8
systolic pressure	693	159	255	70	63	45	1.1	1.1	1.1	1	1.2	1.1	0.5	0.5	0.5	0.6	0.5	0.6	0.3	0.5	0.5	-0.3	0.6	0.8	0.4	-0.03	1	-0.7	0.8	0.9	0.7	0.7	1.2	0.9	1.1	1.3
pulse pressure	692	159	255	70	63	44	0.7	0.7	0.6	0.6	0.8	0.7	0.3	0.3	0.3	0.4	0.4	0.4	0.5	0.7	0.4	0.04	1	0.1	0.9	1.5	0.6	-0.3	2.3	-0.6	1.2	1.7	0.9	0.7	2.6	0.8
cytopenia	686	156	248	72	63	46	0.4	0.4	0.4	0.3	0.4	0.3	0.6	0.6	0.6	0.5	0.7	0.6	1.5	1.7	1.4	1.3	1.9	1.6	2.1	2.4	1.8	0.4	3.3	1.5	3	3.4	2.8	2.1	4.2	2.8


serum creatinine 688 157 248 72 63 47 1.3 1.2 0.5 1.1 0.8 1.3 12.3 8.1 1.5 3.8 2 4.1 22 11.8 10.5 7.6 4.6 6.1 521.8 140.3 126 58.9 22 37.1 522.3 141 126.5 59.5 22.6 37.8
consolidation 694 159 253 72 63 47 0.5 0.5 0.6 0.5 0.3 0.5 0.5 0.5 0.5 0.5 0.5 0.5 -0.1 -0.2 -0.2 -0.2 0.8 -0.1 -1.9 -2 -2 -2 -1.3 -2 2.1 2.2 2.2 2.2 2.2 2.2
grounded glass opacity 694 159 253 72 63 47 0.5 0.5 0.6 0.5 0.3 0.5 0.5 0.5 0.5 0.5 0.5 0.5 -0.1 -0.2 -0.2 -0.2 0.8 -0.1 -2 -2 -2 -2 -1.3 -2 2.2 2.2 2.2 2.2 2.2 2.2
ventilatory support time 700 161 256 72 63 47 -0.03 -0.1 -0.03 0.1 -0.1 -0.03 0.2 0.2 0.3 0.3 0.2 0.3 2.9 3.2 2.8 1.8 4 3.4 8.7 10.5 7.6 3 18.3 11.5 12.1 11.5 11.8 5.5 19 14.4
hospitalization time 700 161 256 72 63 47 0.6 0.5 0.8 0.6 0.9 0.5 1.3 1.2 1.4 1 1.6 1 2 1.9 1.6 1.8 2.3 2.1 4.8 4 2.3 3.8 6.6 4.2 5.6 5 3.3 4.5 7.2 5
platelets 688 156 249 72 63 47 0.4 0.4 0.5 0.5 0.5 0.4 0.5 0.5 0.5 0.5 0.5 0.4 1.3 1 1.6 0.6 1.5 0.7 3.9 2.5 5.7 -0.3 3.7 0.4 4.3 3 6 1.2 4.2 1.3
symptomatic time 700 161 256 72 63 47 0.6 0.5 0.6 0.6 0.7 0.6 0.3 0.3 0.3 0.3 0.4 0.3 1 0.6 0.7 1.2 0.6 1.1 1.8 0.3 0.9 1.9 -0.4 2 2.1 0.9 1.2 2.3 0.9 2.3
indirect bilirubin 686 156 249 71 63 47 0.7 0.5 0.7 0.7 0.9 0.7 0.4 0.3 0.5 0.3 0.6 0.4 3.3 1.7 3.5 1.4 3.6 1.6 19.4 4.1 18.1 1.9 16.3 3.8 19.7 4.5 18.5 2.4 16.7 4.2
direct bilirubin 687 156 250 71 63 47 0.8 0.5 0.8 0.9 1 0.8 0.9 0.3 1 0.7 1.9 0.4 8.1 2.2 4.5 2.8 6.5 1.1 100.3 7.2 22.7 10.2 45.1 0.5 100.6 7.6 23.2 10.6 45.6 1.3
epicardial fat 700 161 256 72 63 47 2.1 1.4 2.3 2.7 2.7 2.8 1 0.8 0.8 1 1.2 1.1 1.6 6.7 0.2 0.2 2.5 0.7 8 67.6 0.1 -0.6 12.2 0.2 8.2 67.9 0.4 0.7 12.5 0.8
creatinine kinase 654 150 237 70 61 44 1 0.5 1.3 1.6 1.1 1.1 1.6 0.7 2.2 1.9 1 1.2 5.4 2.7 5.1 2.1 1.4 2 46.1 9.1 35.6 4.3 1.6 4.4 46.5 9.5 36.1 5 2.3 5
alanine transaminase 688 156 250 71 63 47 1.6 1.4 1.6 2.5 1.5 1.8 3 2.9 1.4 6.6 2 4.1 11.4 10 2.7 6.7 4.1 6.2 164.4 111.3 9.5 47.4 18.9 37.8 164.8 111.8 9.9 48 19.4 38.3
aspartate aminotransferase 688 156 250 71 63 47 1.9 1.3 1.6 4.1 2.1 2.4 4.9 1.6 1.3 13.8 3.1 5 16.1 6.1 3.3 6.3 4.7 6.2 308.6 44 15.6 40.7 24.2 38 309 44.4 16 41.3 24.7 38.6
ferritin 677 154 243 71 62 47 4.4 2.3 4.9 7.4 4.9 6.2 6.2 6.8 3.5 8.8 3.9 11.7 8 10.9 1.6 4.6 1.5 6 88.7 126.1 4.3 27.1 3.3 36.6 89 126.6 4.6 27.5 3.7 37.1
lactic dehydrogenase 700 161 256 72 63 47 1.3 0.9 1.2 2.3 1.3 2 1.3 0.6 0.9 2.8 0.6 1.7 7 0.8 1.9 4.4 0.7 4.9 79.6 1.9 10.4 20.8 0.1 27.7 79.9 2.2 10.6 21.3 0.8 28.1
respiratory rate 663 160 245 58 60 40 0.9 0.8 0.8 1.1 0.9 1 0.4 0.5 0.3 0.3 0.3 0.3 3.9 4.1 0.5 -0.5 0.3 -0.5 30.4 23.2 -0.6 -1 -0.8 0.3 30.7 23.5 0.8 1.1 1 0.7
pulmonary involvement 700 161 256 72 63 47 2.3 2 2.2 2.7 2.3 2.9 0.8 0.8 0.7 0.6 0.7 0.3 -0.7 -0.3 -0.6 -2 -0.6 -2.9 -0.4 -0.7 -0.3 2.8 -0.9 6.4 0.9 0.8 0.7 3.4 1.1 7
fibrinogen 635 144 227 70 59 41 2.1 1.8 2.2 2.4 2 2.5 0.8 0.7 0.8 0.8 0.8 0.6 0.1 0.6 -0.2 -0.4 -0.1 0.1 -0.8 -0.1 -0.7 -0.6 -1.1 -1.1 0.9 0.7 0.8 0.8 1.2 1.1
C-reactive protein 700 161 256 72 63 47 5.7 3.8 4.8 10 5.4 12.3 13 2.9 4.7 28.7 3.2 31.6 16.2 0.8 6 8 0.2 6.4 285.6 -0.1 65.2 63.2 -1 39.5 286 1.1 65.4 63.8 1.2 40.1
neutrophils-lymphocytes ratio 658 145 243 68 60 46 4 2.4 3.4 5.9 4.3 7.5 4.9 2.5 3.8 6.4 4.1 8 3.9 2.5 3.8 2.6 2 3.2 22 7.4 20 7.8 4.5 13 22.4 7.9 20.3 8.3 5 13.5
leukocytes 688 156 249 72 63 47 0.8 0.5 0.7 1.1 0.7 1.1 0.7 0.8 0.6 0.8 0.6 0.9 2.9 7.3 1.5 0.9 1.2 1.1 20.3 70.7 2.9 1.6 1 0.3 20.5 71 3.4 2 1.8 1.4
total neutrophils 700 161 256 72 63 47 0.7 0.5 0.6 1.1 0.7 1.1 0.8 0.9 0.7 0.9 0.7 1 2.6 6.6 1.1 0.6 0.9 0.9 17.5 63.5 2.3 1.2 0.6 0.2 17.8 63.8 2.8 1.6 1.5 1.3
triglycerides 455 108 146 57 40 36 1.2 1.1 1.2 1.3 1.1 1.2 0.8 0.6 0.8 0.7 0.7 1.2 2.8 1.3 2.2 1.5 1.8 3.7 12.7 1.9 6.2 2.4 4.7 15.4 13 2.4 6.6 2.9 5.1 15.8
TyG index 453 108 145 57 40 35 1.5 1.4 1.5 1.8 1.5 1.7 0.5 0.5 0.5 0.6 0.6 0.6 0.9 0.7 0.9 0.8 1.1 0.7 0.7 0.2 1.5 -0.5 1.6 -0.6 1.2 0.8 1.8 1 2 1
glucose 687 157 248 72 63 46 2.8 2.1 2.2 4.3 3 4.9 3 2.5 2.4 3.9 2.9 4.8 2.4 2.6 3.2 1.3 2.3 1.6 6.6 7.1 14.2 0.9 6 1.5 7.1 7.6 14.6 1.8 6.5 2.4
blood urea nitrogen 688 157 248 72 63 47 0.7 0.3 0.5 1.1 1.2 1.6 1.1 0.6 0.8 1.5 1.5 1.2 4.1 4 5.9 4.8 3.4 1.2 26.7 22.6 54 28.7 13.4 1.2 27.1 23 54.3 29.1 13.9 1.9


BUN to creatinine ratio 688 157 248 72 63 47 0.4 0.4 0.3 0.4 0.5 0.6 0.4 0.5 0.3 0.3 0.3 0.3 5 7.4 1.5 1.2 0.8 0.2 63.5 73.8 4.3 1.9 0.3 0.1 63.7 74.1 4.7 2.4 1 0.6
D dimer 676 154 247 69 61 46 4.3 2.5 3.9 8.6 2.7 8.1 15.5 7.2 18.3 27 2.5 17.7 10 8.9 10 5.6 1.8 3.7 113.7 89.4 104 34.7 2.9 14.1 114.2 89.8 104.5 35.3 3.5 14.7
ultrasensitive troponin 627 141 222 66 56 46 2 0.5 0.8 2.4 2.2 2.6 11.9 1.6 7.1 5.8 14.5 6.2 9.8 9 14.1 4 7.1 3.8 102.9 88.3 203.7 18.7 49.1 16.2 103.6 88.9 204.4 19.3 50 16.9
age 700 161 256 72 63 47 0.8 0.6 0.6 0.7 1.1 1.2 0.3 0.2 0.2 0.2 0.1 0.2 0.1 -0.3 -0.4 -0.8 1.2 1.3 -0.4 -0.9 -0.7 0.4 1.2 1.8 0.6 1.1 0.9 0.9 1.7 2.2
right main pulmonary artery 700 161 256 72 63 47 1.2 1 1.2 1.4 1.4 1.6 0.4 0.3 0.4 0.3 0.4 0.4 0.3 0.3 0.5 -0.4 0.3 -0.5 0.1 -0.3 0.5 1.2 0.1 -0.6 0.5 0.5 0.8 1.3 0.5 0.8
left main pulmonary artery 700 161 256 72 63 47 0.9 0.8 0.9 1.1 1.1 1.2 0.3 0.2 0.3 0.2 0.3 0.3 0.5 0.2 0.5 0.1 0.7 0.1 0.7 0.4 0.02 0.5 1.2 -0.6 0.9 0.6 0.6 0.6 1.4 0.7
pulmonary artery main trunk 700 161 256 72 63 47 1 1 0.9 1.1 1 1.1 0.2 0.2 0.2 0.2 0.3 0.3 0.7 0.5 0.3 0.2 0.9 -0.3 1.8 1 0.2 -0.3 1.1 2.9 1.9 1.1 0.4 0.4 1.4 3

4 Supplementary Table 4

ID	variable	Relation with COVID-19	Indicator	Mechanism
M24	Albumin	- Biomarker on admission for risk of death (Tian et al., 2020)	- Decreased levels of albumin (−3.7 g/L, 95% CI, −5.3 to −2.1; P < .00001) (Tian et al., 2020)	
		-Decreased albumin level in the severe group (Ghahramani et al., 2020; Gong et al., 2020; Huang et al., 2020b; Youssef et al., 2020; Küçükceran et al., 2021; Liu et al., 2021a)	- Marked hypoalbuminemia occurred in 38.2%, 71.2%, and 82.4% patients in non-critically ill, critically ill, and death groups, respectively, on admission and 45.9%, 77.7%, and 95.6% of these three groups, respectively, during hospitalization. (Huang et al., 2020b)	
			- Compared to the mild patients, the level of albumin was lower in the severe patients (36 [33-38.5] vs 49.9 [37.4-43.6]; P < .0001) (Wan et al., 2020)	
O2	oxygen saturation	- Hypoxemia as predictor for patient is at risk of requiring admission to the intensive care unit (ICU) (Xie et al., 2020a)	- A concomitant decrease in SpO2, median in the Covid-19 cohort dropped below 93% one day prior to the event (Pimentel et al., 2020)	-Arterial hypoxemia early in SARS-CoV-2 infection is primarily caused by V/Q mismatch and thus persistence of pulmonary arterial blood flow to non-ventilated alveoli, reflected by a marked increase in P(A-a) O2 gradient (Gattinoni et al., 2020)
		- Hypoxemia has such an impact on prognosis and timely treatment decisions (Dhont et al., 2020)		The persistence of high pulmonary blood flow to non-aerated lung alveoli appears to be caused by the relative failure of the hypoxic pulmonary vasoconstriction mechanism (constriction of small intrapulmonary
		- Significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a)		

		- Oxygen saturation among the most important predictors (Incerti et al., 2021)		arteries in response to alveolar hypoxia) during SARS-CoV-2 infection (Lang et al., 2020)
		- Low oxygen saturation in ARDS course of severe and critical COVID-19 types (Ghahramani et al., 2020)		
		- SpO2 and respiration rate were consistently selected as predictive features across outcomes and modeling methods (Wang et al., 2021a)		
		- Patients of ICU Group had lower oxygen saturation (Carlino et al., 2020)		
O3	PAO_2FIO_2 arterial oxygen pressure/ inspired fraction of oxygen	- Oxygen saturation to fraction of inspired oxygen ratio (SpO2/FiO2) as potential predictor of poor outcome for COVID-19 (Lu et al., 2020)	- A strong and significant association between the square root SpO2/FiO2 value and the risk for death, with a unit decrease in the marker corresponding to 1.82-fold increase in the mortality risk (95% CI: 1.56–2.13) (Lu et al., 2020)	
		-Difference in PaO2/FiO2 ratio between survivors and non-survivors indicates this is associated with the severity of illness and thus prognosis (Yang et al., 2020)		
DM1	Estimated glomerular filtration rate (eGFR)	- Reduction of eGFR may be an indicator of disease severity in COVID-19 (Pelayo et al., 2020) (Ouyang et al., 2020)	- Nearly half (49.3%) of the patients with COVID-19 had AKI. Patients with AKI had a significantly lower baseline estimated glomerular filtration rate (eGFR) and higher FiO2 requirement and D-dimer levels on admission (Pelayo et al., 2020)	-Heart-kidney crosstalk could be contributory to this observation, as cardiomyopathy can lead to renal congestion, hypotension, and renal hypoperfusion leading to a reduction in GFR. The myocardial dysfunction can possibly be attributed to hypoxia, thrombotic events, direct viral damage, and cytokine storm in critically ill patients with severe COVID-19 (Pelayo et al., 2020)
		- Lower eGFRrate was associated with an increased hazard of death (Berenguer et al., 2020)		

		- Lymphopenia indicator of propensity to ICU (Huang et al., 2020a; Wang et al., 2020a)	-Mean (IQR) ICU patients: 0.8 (0.5-0.9) x109/L (p= 0.03)	
		- ↑Lymphopenia prevalence in Non-Survivor patients. (p= 0.0001) (Zhou et al., 2020a)	Mean (IQR) Non-ICU patients: 0.9 (0.6-1.2) x109/L (p= 0.03)	
		- Lymphopenia high prevalence in patients with ARDS. (p= <0.001) (Wu et al., 2020a)	(Wang et al., 2020a)	
		- Significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a)	-Mean (IQR) ICU patients: 0.4 (0.2–0.8) x109/L (p= 0.0041)	
H3	Lymphocytes	-Lower levels of lymphocytes count than mild severe patients (P < 0.05) (Ren et al., 2020)	Mean (IQR) Non-ICU patients: 1.0 (0.7–1.1) x109/L (p= 0.0041) (Huang et al., 2020a)	For patients with ARDS who died, lymphocyte counts (difference, −0.23 ×109/L; 95% CI, −0.41 to −0.07 ×109/L; P = .004) and CD8 T cells (difference, −134 cells/μL; 95% CI, −221 to −10 cells/μL; P = .05) were significantly decreased compared with patients with ARDS who survived. (Wu et al., 2020a)
			-Mean (IQR) of Non-Survivor patients: 0.6 (0.5–0.8) x109/L	
			(p= 0.0001). Mean (IQR) of Survivor patients: 1.1 (0.8–1.5) x109/L (p= 0.0001) (Zhou et al., 2020a)	
			- Lymphopenia (lymphocyte count, 0.8 × 109/L [interquartile range {IQR}, 0.6-1.1]) occurred in 97 patients (70.3%) (Wang et al., 2020a)	
			-Mean (IQR) of patients without ARDS 1.08 (0.72-1.45) x109/L. Mean (IQR) of patients with ARDS 0.67 (0.49-0.99) x109/L.	
			(p= <0.001) (Wu et al., 2020a)	
			- Patients requiring hospital admission also had a higher incidence of bilateral	

			infiltrates on chest radiographs (182 patients [54.7%] vs 5 patients [8.5%]), more pronounced lymphopenia (median lymphocyte count, 0.8 cells/μL [IQR, 0.6-1.1 cells/μL] vs 1.0 cells/μL [IQR, 0.7-1.6 cells/μL] (Suleyman et al., 2020)	
			- Lymphocyte counts of the severe patients (median = 0.8 × 109/L) were significantly lower than that of the mild patients (median = 1.2 × 109/L) (Wan et al., 2020)	
			- Lymphopenia was common in 75.4% of the patients (Zhang et al., 2020c)	
CT9	thoracic subcutaneous adipose tissue	- Subcutaneous adipose tissue attenuation do not progress with the severity in COVID-19 (Iacobellis et al., 2020a)		
DM7	TyG-BMI index	- Risk factor (Albashir, 2020)	- Overweight patients were associated with an 86% higher risk, and obesity with a 142% higher risk, of developing severe pneumonia compared with normal weight patients (Albashir, 2020)	The highest BMI was seen more often in serious cases and non-survivors. (Peng et al., 2020)
DB1	body mass index (BMI)	- BMI >40 kg/m2 was the second strongest independent predictor of hospitalization (Bhasin et al., 2020; Petrilli et al., 2020)	- Random-effects dose-response meta-analysis showed a linear association between BMI and both severe COVID-19 and mortality (Du et al., 2021)	
		- BMI was associated strongly with risk of death related to COVID-19(Sattar et al., 2020)		
		- Obesity (BMI > 30 kg/m2) was associated with a significantly increased risk of critical COVID-19 and mortality (Du et al., 2021)		
B0	weight	- Associated with BMI		

		(ICNARC – Intensive Care National Audit & Research Centre)		
CT13	presence of steatosis on tomography	- The liver tissue showed moderate microvesicular steatosis and mild lobular activity, but there was no conclusive evidence to support SARS-CoV-2 infection or drug-induced liver injury as the cause (Xu et al., 2020). -Presence of fatty liver is a strong predictor for severe disease. (Medeiros et al., 2020; Parlak et al., 2021)		Novel coronavirus may produce, in some cases, relevant hepatic damage, probably through the immune interactions requiring the action of intrahepatic cytotoxic T cells and Kupffer cell (Zhang et al., 2020c). Direct cytotoxicity due to active viral replication in hepatic cells: SARS-CoV-2 binds to target cells through ACE2. Because ACE2 is expressed abundantly in the liver and in particular on biliary epithelial cells, the liver is a potential target for direct infection (Amin, 2021)
HR	heart rate	- Cardiac arrhythmia (Kang et al., 2020) - Patients of ICU group had higher heart rate (Carlino et al., 2020)	- ≥125 beats per min, was present in 4% of Non-Survivor patients. (p=0.024) (Zhou et al., 2020a)	
T0	axillary temperature	- Main clinical symptom of COVID-19 patients - ↑ (Rod et al., 2020) - Higher temperature was related to severe 2019 novel coronavirus pneumonia and composite endpoint. (R1 = 0.362, R2 = 0.383, P3<0.001, P4<0.001) (Zhang et al., 2020b)	- Fever in 88.5% of COVID-19 patients. Body temperature is a risk factor with a high consistency as predictor for COVID-19 severity (Rod et al., 2020)	
M36	Prothrombin	- ↑ indicator of propensity to ICU. (p= 0.012) (Huang et al., 2020a) - ↑ time, in Non-Survivor patients (p= 0.0004) (Zhou et al., 2020a)	- Mean (IQR) ICU patients: 12.2 (11.2-13.4) s (p= 0.012) Mean (IQR) Non-ICU patients: 10.7 (9.8-12.1) s (p= 0.012) (Huang et al., 2020a)	For patients with ARDS who died, coagulation function indices (D-dimer [difference, 2.10 μg/mL; 95% CI, 0.89-5.27 μg/mL; P = .001]) were significantly elevated compared with

		- Coagulation indexes of all of the patients were nearly in the normal range, the Pt, APTT, and d-dimer of the severe patients were higher than those of the mild patients (Wan et al., 2020)	- Mean (IQR) of Non-Survivor patients: 12.1 (11.2-13.7) s	patients with ARDS who survived. (Wu et al., 2020a)
			(p= 0.0004). Mean (IQR) of Survivor patients: 11.4 (10.4-12.6) s (p= 0.0004) (Zhou et al., 2020a)	
			- Mean (IQR) of Non-Survivor patients: 15.5 (14.4-16.3) s (p= <0.001). Mean (IQR) of Survivor patients: 13.6 (13.0-14.3) s (p= <0.001) (Tang et al., 2020)	
			- Baseline characteristics of the first 99 patients hospitalized in Wuhan found that 5% had elevated prothrombin (PT). (Chen et al., 2020b)	
			- Prolonged prothrombin time (13.0 seconds [IQR, 12.3-13.7]) in 80 patients (58%) (Wang et al., 2020a)	
			- Prolonged PT in patients at high risk of severe COVID-19 (Elshazli et al., 2020; Ghahramani et al., 2020; Liao et al., 2020; Liu et al., 2021a)	
M33	vitamin D	- Association between vitamin D deficiency and poor clinical outcome (Raharusun, 2020; Munshi et al., 2021) (Hastie et al., 2020; Radujkovic et al., 2020)		Vitamin D is a key regulator of the renin-angiotensin system that is exploited by SARS-CoV-2 for entry into the host cells. Reduces concentration of pro- inflammatory cytokines and increases levels of anti-inflammatory cytokines, enhances the production of natural antimicrobial peptide and activates defensive cells such as macrophages that could destroy SARS-CoV-2 (Kumar et al., 2021)

B1	Height	- Associated with BMI (ICNARC – Intensive Care National Audit & Research Centre)		
H8	Hemoglobin	- The severity of disease and prognosis of patients with COVID-19 might depend on lower hemoglobin levels as severe cases had significantly lower hemoglobin levels than moderate cases. (Taneri et al., 2020)	- Severe COVID-19 cases had lower hemoglobin [weighted mean difference (WMD), − 4.08 g/L (95% CI − 5.12; − 3.05)] and red blood cell count [WMD, − 0.16 × 1012 /L (95% CI − 0.31; − 0.014)], and higher ferritin [WMD, − 473.25 ng/mL (95% CI 382.52; 563.98)] and red cell distribution width [WMD, 1.82% (95% CI 0.10; 3.55)] (Taneri et al., 2020). - In severe COVID-19 hematocrit (HCT) was significantly higher in patients with CVD than in non-CVD patients (P=0.007) (Xie et al., 2020c)	Low hemoglobin in COVID-19 patients, especially on populations at risk of complications and mortality, could indicate that the patients could suffer from a decreased capability of hemoglobin to support the increased peripheral tissue demands for oxygen due to the hyper-metabolic states during infection (Taneri et al., 2020)
P1	diastolic blood pressure (DBP)			
DP1	Mean Arterial Pressure (MAN)			
P0	systolic blood pressure (SBP)	- Severe patients had significantly higher value of respiratory rate, systemic blood pressure (SBP), and mortality and/or more likely to receive auxiliary ventilation, and invasive mechanical ventilation (all $P < 0.05$)(Ren et al., 2020)	- Arterial hypertension is risk of death in patients odds ratio [OR], 2.5; 95% confidence interval [CI], 2.1-3.1; P < .00001)	
		- Pearson correlations showed that hypertension and systolic blood pressure (SBP) were associated with death and	Coronary heart disease is risk of death in patients odds ratio (OR, 3.8; 95% CI, 2.1-6.9; P < .00001) (Tian et al., 2020)	

		respiratory distress parameters. SBP but not hypertension was a covariate in both mortality and survival prediction models. SBP was elevated in deceased compared with discharged COVID-19 patients.(Caillon et al., 2021)	- Significant predictors of heart failure were average systolic blood pressure (SBP) (hazard ratio (HR) per 10 mmHg 1.89, 95% confidence interval (CI): 1.15, 3.13) and pulse pressure (HR per 10 mmHg 2.71, 95% CI: 1.39, 5.29). The standard deviations of SBP and diastolic BP were independently associated with mortality and ICU admission. (Ran et al., 2020)	
DP0	pulse pressure (PP)	- Hypertension is a risk factor (Sardu et al., 2020)		Hypertension (Sardu et al., 2020)
		- Risk of death in patients with COVID-19 infection (Sardu et al., 2020).		
H15	Cytopenia	- High prevalence in Non-ICU patients. (p= <0.041) (Huang et al., 2020a)	- White blood cell count less than 4x109/L	Available data of 33 COVID-19 patients who either recovered or died. Characteristics of the 5 nonsurvivors compared with the 28 survivors included rising D-dimer, progressive lymphopenia, and renal dysfunction. (Wang et al., 2020a)
		- High prevalence in survivor patients (p= <0.0001) (Zhou et al., 2020a)	8% of ICU patients	
		- High prevalence in patients without ARDS (p= <0.001) (Wu et al., 2020a)	33% of Non-ICU patients	
			(p= <0.041)	
			-White blood cell count less than 4x109/L	
			9% of Non-Survivor patients	
			20% of Survivor patients	
			(p= <0.0001) (Zhou et al., 2020a)	
			- White blood cell count. Mean (IQR) of patients without ARDS 5.02 (3.37-7.18) x109/L. Mean (IQR) of patients with	

			ARDS 8.32 (5.07-11.20) x109/L. (p= <0.001) (Wu et al., 2020a)	
M10	Creatinine	- ↑ indicator of propensity to ICU. (p= <0.04) (Wang et al., 2020a)	- Mean (IQR) ICU patients: 80 (66-106) µmol/L (p= <0.04)	
		- Severe group had higher levels of creatinine (Ghahramani et al., 2020; Iavarone et al., 2020; Mudatsir et al., 2020; Shao et al., 2020; Malik et al., 2021)	- Mean (IQR) Non-ICU patients: 71 (58-84) µmol/L (p= <0.04) (Wang et al., 2020a)	
			- Severe group had higher levels of aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatinine, uric acid (UA), CRP, and FPG, compared with mild group (P < 0.05)(Ren et al., 2020)	
CT16	consolidation in tomography	- Main sign on CT imagen of COVID-19 lesion (Li and Xia, 2020)	- CT showed consolidation in 49 of the 51 (96.1%) patients	In COVID-19 patients, consolidation may relate to cellular fibromyxoid exudates in alveoli (Xu et al., 2020)
		- This manifestation could serve as an alert in the management of patients (Larici et al., 2020)	CT showed increasing density of consolidation on the first and second follow-up studies, which indicates marked disease progression, and showed findings suggestive of improvement on the third and fourth follow-up studies. Occurrence rate of 2~64% (Li and Xia, 2020)	
			- The majority (134, 99.3%) had abnormal results, showing typical images that were bilateral multiple ground-glass opacities or consolidation (Zhang et al., 2020c)	
CT17	presence of grounded glass (GG) and consolidation		- Enlargement (greater than 3 mm in diameter) of subsegmental pulmonary vessels within the areas of GG has also been described in the early phase of	

	(0=no, 1=yes) (Larici et al., 2020)		COVID-19 pneumonia in up to 89 % of cases. (Larici et al., 2020) (Grassi et al., 2020; Pan et al., 2020b)	
H14	platelets		- Mild thrombocytopenia has been detected in 58–95% of severe cases of COVID-19 (Chen et al., 2020b; Cheng et al., 2020a; He et al., 2020; Hu et al., 2020; Suleyman et al., 2020; Wan et al., 2020; Wu et al., 2020a; Yang et al., 2020; Abrishami et al., 2021; Kim et al., 2021; Wool and Miller, 2021)	Lung injury (Wool and Miller, 2021)
symD	days with symptoms before arrival to hospital	- The mean time from symptom onset to hospitalization overall is 5.74 days, which is slightly longer as compared to the reported delay in other countries, but depending on the patient population, estimates range between 3 and 10.4 days (Faes et al., 2020)	- The time from symptom onset to hospitalization is largest in the working age population (20–60 years), followed by the elderly (60–80) years. (Faes et al., 2020)	
M21	indirect bilirubin	- Increased serum bilirubin level was observed in fatal cases (Wu et al., 2020b)		- Risk factor of coronary heart disease (Deng et al., 2020; Ouyang et al., 2020; Wu et al., 2020a; Lopes et al., 2021; Sun et al., 2021)
M20	direct bilirubin	- Higher direct bilirubin levels is associated with severe Covid-19 (Gong et al., 2020; Liang et al., 2020)	- Patients with severe Covid-19 displayed higher bilirubin levels compared to those with milder forms (mean difference ranging between 0.27 and 0.95 μmol/L) (Paliogiannis et al., 2020) - Bilirubin concentration was significantly higher in patients with severe Covid-19 (SMD: 0.48 μmol/L;	

			95% CI, 0.11 to 0.85 μmol/L, P = .012) (Paliogiannis and Zinellu, 2020)	
CT4	average epicardial fat	- Its elevation increases mortality in COVID-19 (Deng et al., 2020; Iacobellis et al., 2020b, 2020a; Kim and Han, 2020; Malavazos et al., 2020; Zhao, 2020; Abrishami et al., 2021; Bihan et al., 2021; Grodecki et al., 2021; Slipczuk et al., 2021)		Is associated with extent of pneumonia and adverse outcomes (Grodecki et al., 2021)
M26	creatinine kinase	- Elevated during COVID-19, it is a nonspecific marker of muscle damage (Chen et al., 2020b; Deng et al., 2020, 2020; Henry et al., 2020; Huang et al., 2020b; Izcovich et al., 2020; Liu et al., 2020; Mudatsir et al., 2020; Qiu et al., 2020; Rodriguez-Morales et al., 2020; Suleyman et al., 2020; Wang et al., 2020a; Zhang et al., 2020d; Abrishami et al., 2021; Malik et al., 2021)	- Creatine kinase, U/L were 39·0 (19·5–151·0) in non survivors patients versus 18·0 (12·5–52·1) survivors patients p<0.001(Zhou et al., 2020a)	
M23	alanine transaminase (ALT)	- Elevated in COVID (Zhang et al., 2020b) - High values of the pyridoxal phosphate-dependent enzymes AST and ALT were significantly associated with the COVID-19 disease (Ferrari et al., 2020; Ou et al., 2020) - Related to severe coronavirus pneumonia and composite endpoint (Zhang et al., 2020b) -Increase alanine aminotransferase (ALT) in the severe group compared with the non-severe group. (Ghahramani et al., 2020; Huang et al., 2020b; Mudatsir et al.,		

		2020; Youssef et al., 2020; Liu et al., 2021a; Malik et al., 2021)		
M22	aspartate aminotransferase (AST)	- ↑ indicator of propensity to ICU (p= <0.001) (Wang et al., 2020a)	- Mean (IQR) ICU patients: 53 (30-70) U/L (p= <0.001)	For patients with ARDS who died, the value of liver damage indices (total bilirubin [difference, 2.60 μM; 95% CI, 0.30-5.20 μM; P = .03]), were significantly elevated compared with patients with ARDS who survived (Wu et al., 2020a)
		- High levels associated with patients among ARDS. (p= <0.001) (Wu et al., 2020a)	Mean (IQR) Non-ICU patients: 29 (21-38) U/L (p= <0.001) (Wang et al., 2020a)	
		-Increase levels of aspartate aminotransferase (AST) in the severe group compared with the non-severe group. (Ghahramani et al., 2020; Huang et al., 2020b; Lei et al., 2020; Mudatsir et al., 2020; Youssef et al., 2020; Malik et al., 2021; Zhao et al., 2021)	- Mean (IQR) of patients without ARDS 30.0 (24.0-38.50) U/L. Mean (IQR) of patients with ARDS 38.0 (30.5-53.0) U/L. (p= <0.001) (Wu et al., 2020a)	
			-Severe group had higher levels of aspartate aminotransferase (AST), lactate dehydrogenase compared with mild group (P < 0.05) (Ren et al., 2020)	
M34	Ferritin	- Biomarkers, such high serum procalcitonin and ferritin have also emerged as poor prognostic factors in COVID-19 patients (Terpos et al., 2020) (Rostami and Mansouritorghabeh, 2020)	- Indicator of thrombotic complication, mean (min-max) 1182 (697-2081) μg/L (P=0.0020) (Al-Samkari et al., 2020).	-Levels of serum ferritin, d-dimer, lactate dehydrogenase, and IL-6 are increased during the worsening of the disease, providing an indication of the risk of mortality (Zhou et al., 2020a)
			-Severe patients and discharged patients have greater proportions of increased level of ferritin than non-severe patients and hospitalized patients (100% vs. 50%, 92.3% vs. 37.9% respectively, P < .001) and suggested that serum ferritin is a potential risk factor of poor prognosis in COVID-19 patients. (Sun et al., 2020).	-Hyperferritinemia caused by the excessive inflammation due to the infection is associated with the admission to the intensive care unit and high mortality, and represents an indication to recognize high-risk patients to guide the therapeutic intervention to control inflammation (Cheng et al., 2020b)
M25	lactic dehydrogenase	- Severe group had higher levels of lactate dehydrogenase (LDH), compared with mild group (P < 0.05) (Ren et al., 2020)	- Related to severe coronavirus pneumonia and composite endpoint (Zhang et al., 2020b)	

		- Increased lactic dehydrogenase in the severe group (Ghahramani et al., 2020; Gong et al., 2020; Ouyang et al., 2020; Zhou et al., 2020a)	were significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a)	
			- Elevated lactate dehydrogenase (261 U/L [IQR, 182-403]) in 55 patients (39.9%) (Wang et al., 2020a)	
BR	breathing rate	- Significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a)	- Multivariate regression showed increasing odds ratio (OR) of in-hospital death associated with respiratory rates >20 breaths/min (OR: 5.14, 95% CI: 1.19-22.15, p = 0.028) (Vahedian-Azimi et al., 2021)	Chest pain (Andrikopoulou et al., 2020 in pregnant women)
		- SpO2 and respiration rate were consistently selected as predictive features across outcomes and modeling methods (Wang et al., 2021a)	- Severe patients had significantly higher value of respiratory rate, systemic blood pressure (SBP), and mortality and/or more likely to receive auxiliary ventilation, and invasive mechanical ventilation (all $P < 0.05$)(Ren et al., 2020)	
		- >24 breaths per minute is associated with severe cases in COVID-19 (Huang et al., 2020b; Pan et al., 2020a; Ren et al., 2020; Suleyman et al., 2020; Wan et al., 2020; Zhang et al., 2020c)		(Andrikopoulou et al., 2020; Huang et al., 2020b; Pan et al., 2020a; Ren et al., 2020; Suleyman et al., 2020; Wan et al., 2020; Wang et al., 2020a; Zhang et al., 2020b, 2020a)
M37	Fibrinogen	- Increased during COVID-19 (Connors and Levy, 2020) (Ghahramani et al., 2020; Ouyang et al., 2020)	- Fibrinogen levels in all patients were elevated on admission.	-The coagulopathy with COVID-19 is a result of the inflammatory response to SARS-CoV-2 infection resulting in thrombo inflammation and driving thrombosis.
		- Decreased in disseminated intravascular coagulation (Connors and Levy, 2020)	- Nonsurvivors had evidence of progressive disseminated intravascular coagulation with decreased fibrinogen (Connors and Levy, 2020)	-Coagulopathy is manifest as elevated fibrinogen, elevated D-dimers, and minimal change in PT, aPTT, and platelet count in early stages of infection (Ranucci et al., 2020)
				-IL-6 is a powerful pro-inflammatory cytokine, which induces tissue factor

				gene expression in endothelial cells and monocytes, fibrinogen synthesis, and platelet production, without affecting fibrinolysis.
				(Xie et al., 2020b)
M13	C-reactive protein (PCR)	- Elevated hs-CRP can effectively triage suspected COVID-19 patients (Li et al., 2020; Mardani et al., 2020)	- CRP level at admission represent a simple and independent factor that can be useful for early detection of severity during COVID-19 (Ahnach et al., 2020)	Hypercoagulability is due to the profound derangement of hemostasis and is the likely contributor to pulmonary embolism and/or deep vein thrombosis of the lower limbs observed in patients with COVID-19 (Panigada et al., 2020).
		-Higher concentrations of C-reactive protein was associated with an increased hazard of death (Berenguer et al., 2020) (Gong et al., 2020)	-Significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a)	The increase in inflammation markers and acute phase reactants are linked with the underlying systemic vasculitic processes and the cytokine storm that cause most parenchymal lesions in vital organs (Ahmed and Ghani, 2020).
		- CRP was positively correlated to the severity of COVID-19 pneumonia (Chen et al., 2020c)	- Severe group had higher levels of aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatinine, uric acid (UA), CRP, and FPG, compared with mild group ($P < 0.05$) (Ren et al., 2020)	
		- Indexes related to myocardial injury, such as creatine kinase, glutamic oxaloacetylase, LDH, and C-reactive protein (CRP), increased more significantly in the severe patients (Wan et al., 2020)	- Higher concentration of C-reactive protein (91.9%) in severe patients vs non severe ones ($p<0.001$) (Zhang et al., 2020c)	
DH6				

	neutrophils-lymphocytes relation (NLR)	- NLR can serve as a predictor of COVID-19 disease severity (Wang et al., 2021b) (Fu et al., 2020; Ghahramani et al., 2020; Lagunas-Rangel, 2020; Liang et al., 2020; Ouyang et al., 2020; Ok et al., 2021)		A statistically significant positive correlation is present between PCT and NLR in the severe group at the first week of admission (Wang et al., 2021b)
H0	Leukocytes	- Related to severe coronavirus pneumonia and composite endpoint (Zhang et al., 2020b) - Severe patients had higher values (Ren et al., 2020) (Ok et al., 2021)		
H1	total neutrophils	- Related to severe coronavirus pneumonia and composite endpoint (Zhang et al., 2020b) - Significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a) - Severe patients had higher values (Ren et al., 2020) (Elshazli et al., 2020; Izcovich et al., 2020; Ouyang et al., 2020; Ok et al., 2021)	- Increased in COVID-19, it is found in current infection (Deng et al., 2020; Huang et al., 2020b; Hu et al., 2020; Qin et al., 2020; Shi et al., 2020; Wan et al., 2020; Wu et al., 2020a; Zhou et al., 2020a; Abrishami et al., 2021)	
M1	triglycerides	- Risk factor for atherosclerotic cardiovascular disease, elevated during COVID-19, excessive free fatty acids release (Aung et al., 2020; Deng et al., 2020; Huang et al., 2020b; Prilutskiy et al., 2020; Ren et al., 2020; Wang et al., 2020b; Webb et al., 2020; Saand et al., 2021)		It is related to atherosclerotic cardiovascular disease, acute pancreatitis (Peng et al., 2020)

DM6	TyG index	- TyG index was significantly associated with an increased risk of severe case and mortality (Ren et al., 2020).	- TyG index levels were significantly higher in the severe cases and death group (mild vs. severe 8.7 ± 0.6 vs. 9.2 ± 0.6, $P < 0.001$; survivor vs. deceased 8.8 ± 0.6 vs. 9.3 ± 0.7, $P < 0.001$), respectively (Ren et al., 2020). - TyG index, which calculated by triglycerides and glucose, was markedly higher in severe cases than in mild ones ($P < 0.05$) (Ren et al., 2020).	
M5	glucose	-Increased glucose level in the severe group compared with the non-severe group (Ghahramani et al., 2020)	- Severe COVID-19 was associated with higher blood glucose (WMD 2.21, 95% CI: 1.30-3.13, $P < 0.001$) (Chen et al., 2020a) - Glucose was significantly increased ($P < 0.05$) in non-survivors compared with survivors (Ouyang et al., 2020)	Hyperinsulinaemia, hyperglycaemia and hypertension increase inflammation, coagulation and thrombosis risk (Cooper et al., 2020)
M8	BUN	- It can be used to predict renal function, higher in severe cases (Chen et al., 2020b; Cheng et al., 2020a; Ghahramani et al., 2020; Gong et al., 2020; Henry et al., 2020; Izcovich et al., 2020; Liu et al., 2020; Mudatsir et al., 2020; Qiu et al., 2020; Webb et al., 2020; Abrishami et al., 2021; Küçükceran et al., 2021; Ok et al., 2021; Zhao et al., 2021)	- Multivariate regression showed increasing odds ratio (OR) of in-hospital death associated with blood urea nitrogen (BUN) >19 mg/dL (OR: 4.54, 95% CI: 1.30-15.85, $p = 0.017$) (Vahedian-Azimi et al., 2021)	It is a marker for predicting persistent organ failure after 48 h of hospital admission; associated to renal failure, heart failure, pulmonary embolism, acute pancreatitis, ventilation failure, diabetes mellitus (Cheng et al., 2020a; Ghahramani et al., 2020; Qiu et al., 2020; Abrishami et al., 2021)
DM4	BUN to creatinine ratio	- Blood urea nitrogen-to-creatinine ratio (BCR) is a predictor for mortality in patients with COVID-19. (Liu et al., 2021b)	- In multivariate Cox proportional hazard model BUN/Cr ratio (hazard ratio [HR] = 1.02; 95% CI: 1.01-1.05; $P = .030$) was independent predictors for survival of COVID-19 disease. (Ok et al., 2021)	

M35	dimer D	- Were significantly correlated with death events identified using bivariate logistic regression (Pan et al., 2020a) - Coagulation indexes of all of the patients were nearly in the normal range, the Pt, APTT, and d-dimer of the severe patients were higher than those of the mild patients (Wan et al., 2020) - Increase of D-dimer in severe Covid-19 group (Ghahramani et al., 2020; Ouyang et al., 2020; Zhou et al., 2020a; Abderrahim et al., 2021)	- A biomarker of fibrin formation and degradation. Coagulopathy disorder indicator. It highly correlates with a poor prognosis when increased (Chen et al., 2020b; Cheng et al., 2020a; Connors and Levy, 2020; Deng et al., 2020; Gao et al., 2020; He et al., 2020; Huang et al., 2020a; Qiu et al., 2020; Suleyman et al., 2020; Wan et al., 2020; Zhang et al., 2020c; Lenka et al., 2021)	It is correlated with venous thromboembolism, cardiac injury and sustained inflammatory response (Breakey and Escher, 2020; Cheng et al., 2020a; Deng et al., 2020; Lippi and Plebani, 2020; Zhang et al., 2020c)
M29	Ultrasensitive troponin	- Elevated troponin is an independent predictor of 30-day mortality (García de Guadiana-Romualdo et al., 2021)	- Sex-specific elevated troponin levels were significantly associated with 30-day mortality, with adjusted odds ratios (ORs) of 3.00 for total population, 3.20 for cardiac troponin T and 3.69 for cardiac troponin I.(García de Guadiana-Romualdo et al., 2021)	
		- Associated to a worse prognosis in hospitalized patients with severe COVD-19 (Gómez-Mesa et al., 2021) - Higher levels were observed in death events group (Pan et al., 2020a) (Mudatsir et al., 2020)	- Hypersensitive cardiac troponin I(hs-cTnI) > in COVID-10 patients (Zheng et al., 2020)	
D1	age	- Aging is an important risk factor for severe COVID-19 disease and its adverse health outcomes including hospitalization, ICU admission, and death (Wu et al., 2020a)	- Case fatality ratio (CFR) of COVID-19 increases with age, from 0.4 % or lower in patients aged in the 40s or younger, 1.3 % among those in their 50s, 3.6 % in their 60s, 8% in their 70s, to 14.8 % in	An immune hypothesis for COVID-19 vulnerability of older adults. It involves age-related impairment of immune defense against SARS-CoV-2 infection, or immunosenescence,

			their 80s or older; the overall CFR is 2.3 %(Wu et al., 2020a; Zhu et al., 2020)	and increased risk for immunopathology (Sardu et al., 2020)
		- The risk of fatal or critical care unit-treated COVID-19 increased with age(McGurnaghan et al., 2021)	-Non-survivors tend to be older (median age 80 vs. 64) (Bertsimas et al., 2020)	
		- Age was the most important predictor of all-cause mortality (Incerti et al., 2021)	- Increasing odds of in-hospital death associated with older age (odds ratio 1·10, 95% CI 1·03–1·17, per year increase; p=0·0043) (Zhou et al., 2020a)	
		- Elder age was an independent risk factors for death of severe patients (Zhang et al., 2021)	- Compared to mild patients, severe patients were significantly older (median age 56 years [IQR, 52-73] vs 44 years [IQR, 33-49]; P < .001) (Wan et al., 2020)	
			-Log-linear relationship between age and risk of death continues into older age groups(O'Driscoll et al., 2021)	
CT6	right main artery diameter	No items found.		
CT7	left main artery diameter	No items found.		
CT5	main trunk pulmonary artery diameter	-Increased main pulmonary artery diameter is associated with poorer prognosis for patients with COVID-19 pneumonia.(Grassi et al., 2020; Raoufi et al., 2020; Erdoğan et al., 2021; Esposito et al., 2021; Jalde et al., 2021; Yildiz et al., 2021)	- Increased in 8/126 (6.3%) patients. (Grassi et al., 2020)	Discrete pulmonary nodules, increased trunk diameter of the pulmonary artery, pleural effusion can be found but in a low non-significant percentage of cases (7.9%, 6.3%, 14.3%, respectively) (Grassi et al., 2020)

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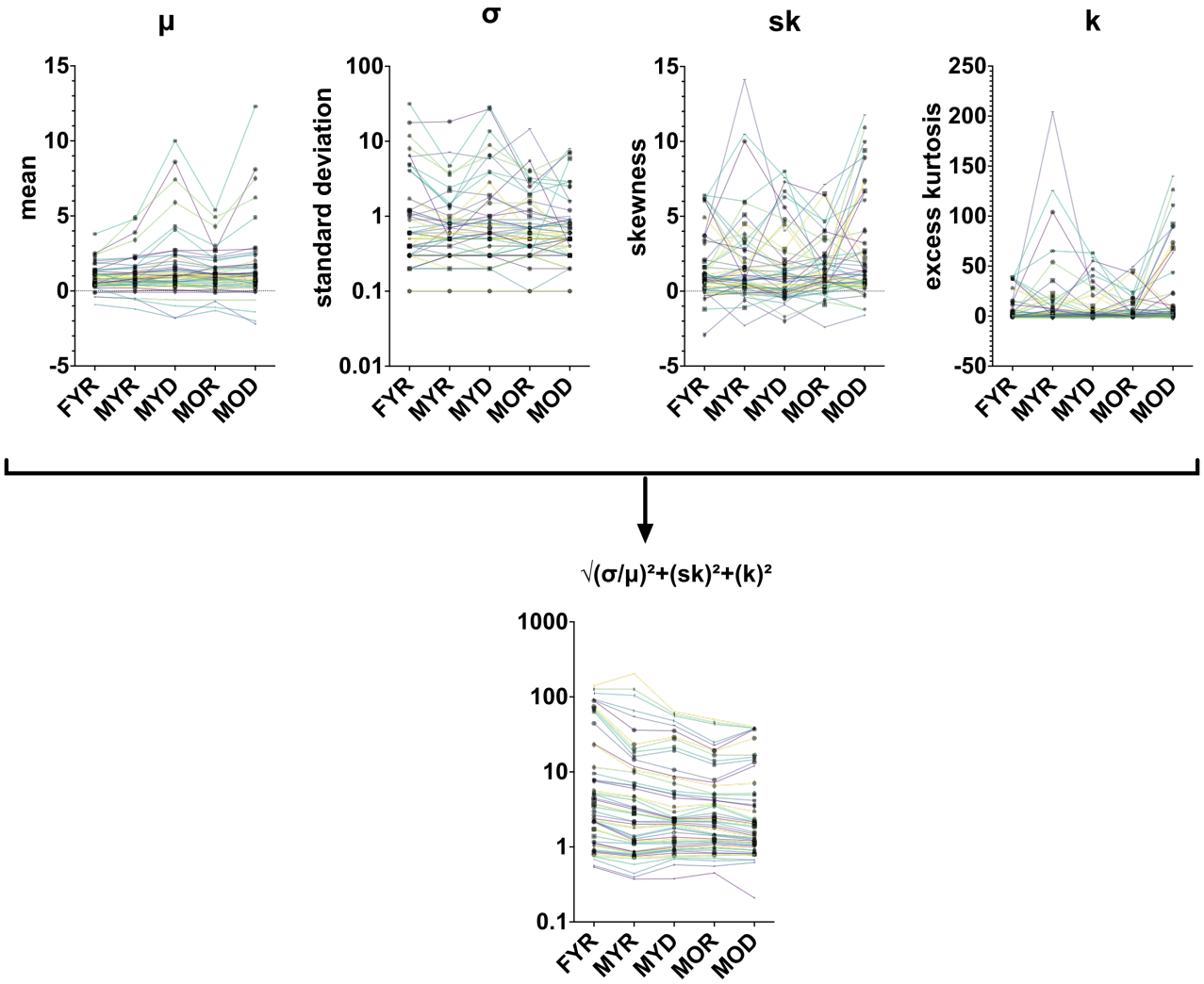
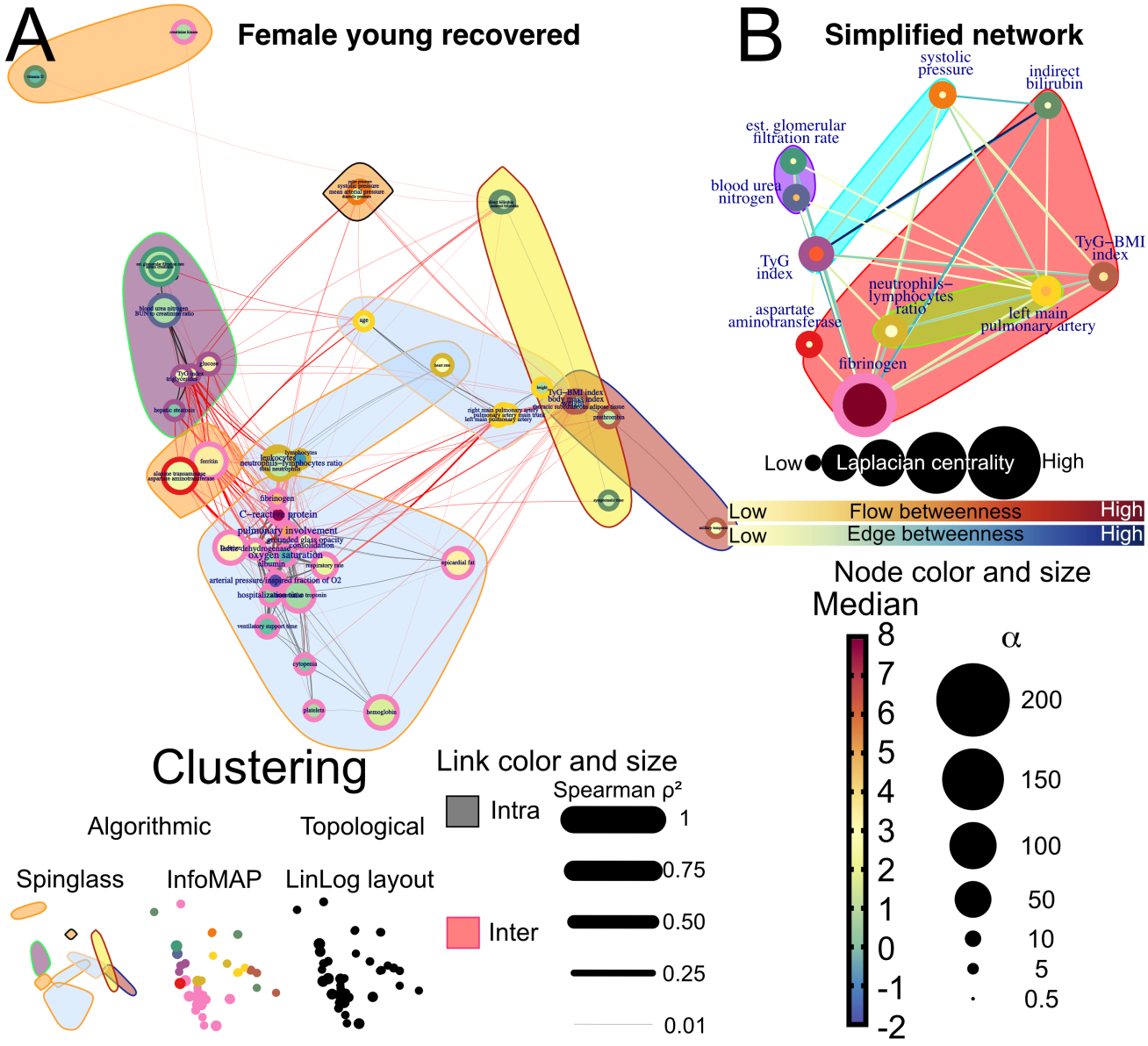
5 Supplementary Figure 1



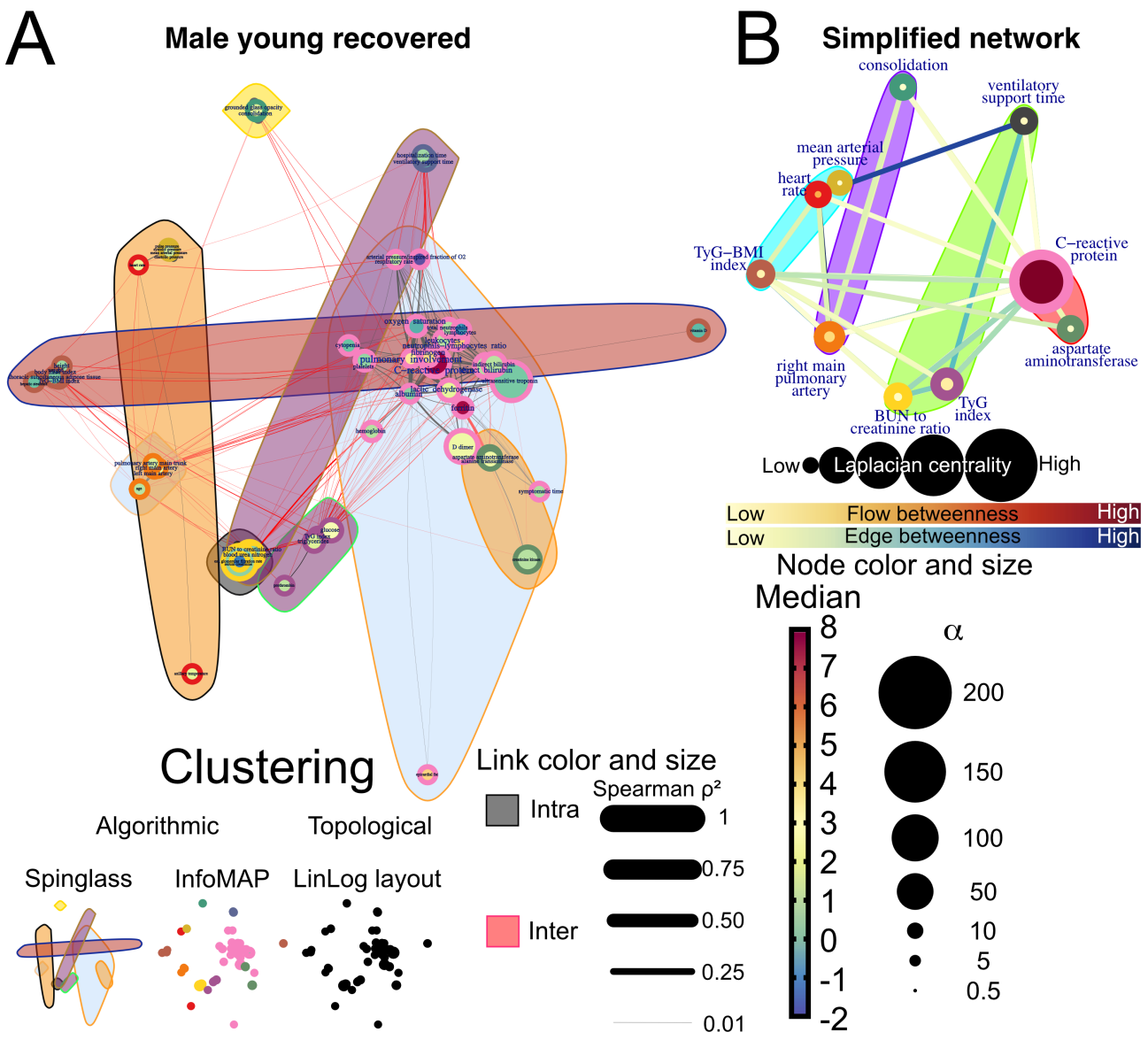
Supplementary Figure 1. Descriptive statistics behavior of each group. The four moments of the distribution of each physiological variable are plotted for each group and summarized by α.

6 Supplementary Figure 2



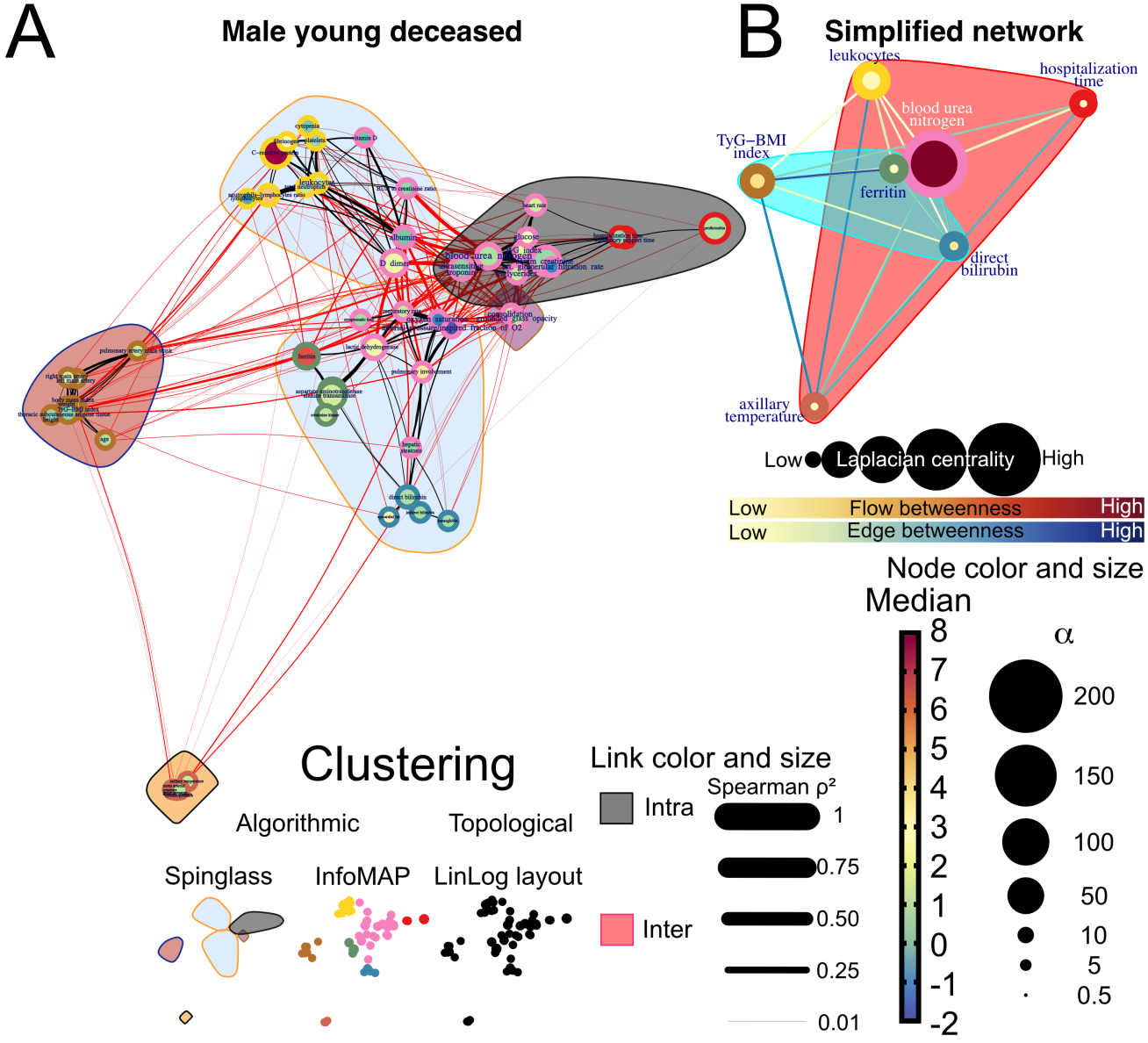
Supplementary Figure 2. Physiological network in female young recovered group. (A) The five study groups' networks were plotted. Links between nodes in the same cluster are shown in black, while shared links are shown in red. Each correlation's strength is indicated by the width of the links. LinLog layout was used to arrange the nodes. The color of the node represents the normalized median value for each study group, and the size represents the deviation from the normal distribution. The color-shaded areas in this network represent spinglass clusters, while the node border represents the nodes that will be collapsed into a single supernode by InfoMAP clustering. (B) depicts a simplified network in which supernodes are labeled with the name of the physiological variable with the greatest influence within the InfoMAP community. Edges were kept, resulting in a network with multiple edges. The color of the edge represents its betweenness. The flow betweenness centrality is represented by node color, and the laplacian centrality is represented by node size.

7 Supplementary Figure 3



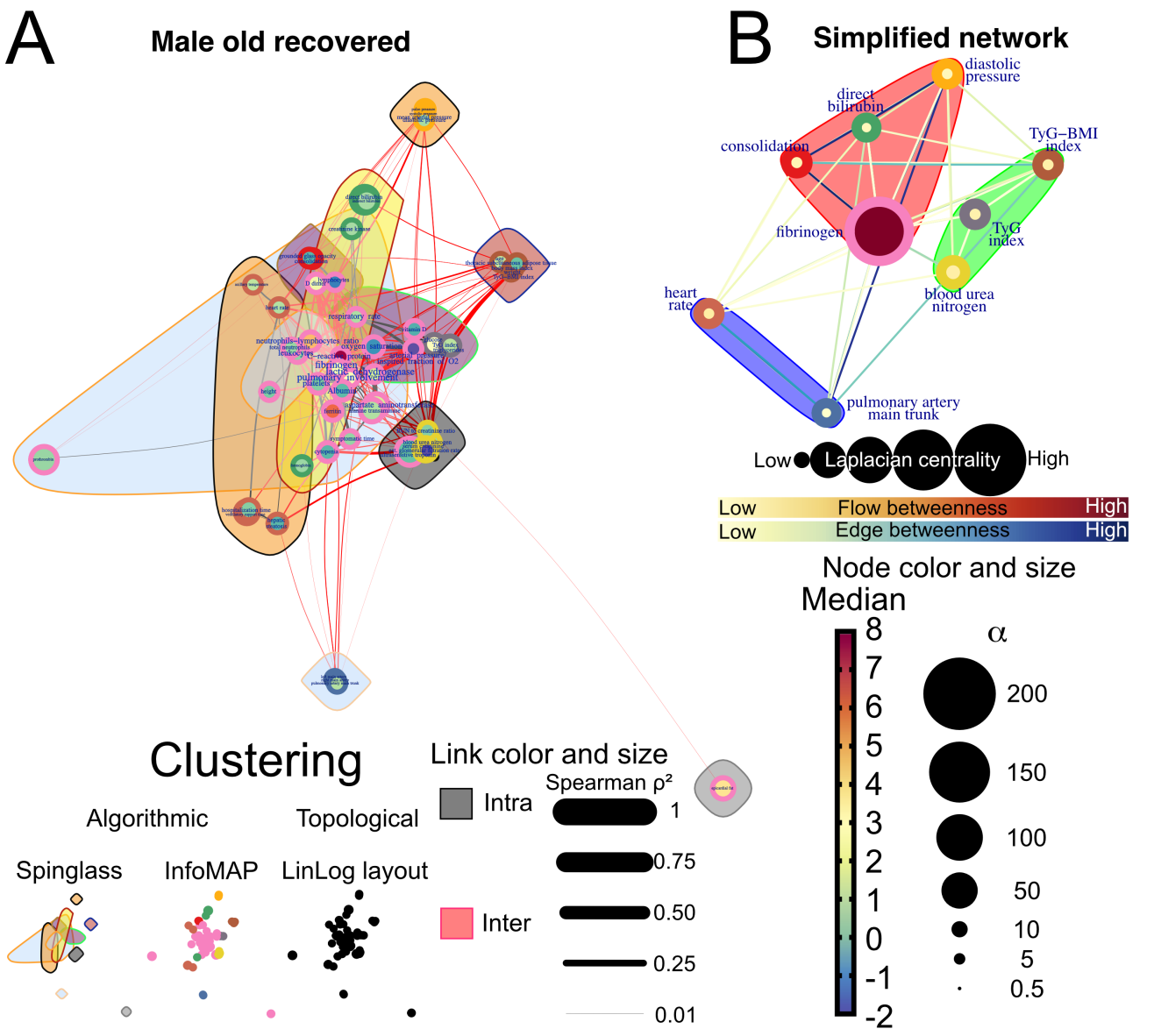
Supplementary Figure 3. Physiological network in male young recovered group. (A) The five study groups' networks were plotted. Links between nodes in the same cluster are shown in black, while shared links are shown in red. Each correlation's strength is indicated by the width of the links. LinLog layout was used to arrange the nodes. The color of the node represents the normalized median value for each study group, and the size represents the deviation from the normal distribution. The color-shaded areas in this network represent spinglass clusters, while the node border represents the nodes that will be collapsed into a single supernode by InfoMAP clustering. (B) depicts a simplified network in which supernodes are labeled with the name of the physiological variable with the greatest influence within the InfoMAP community. Edges were kept, resulting in a network with multiple edges. The color of the edge represents its betweenness. The flow betweenness centrality is represented by node color, and the laplacian centrality is represented by node size.

8 Supplementary Figure 4



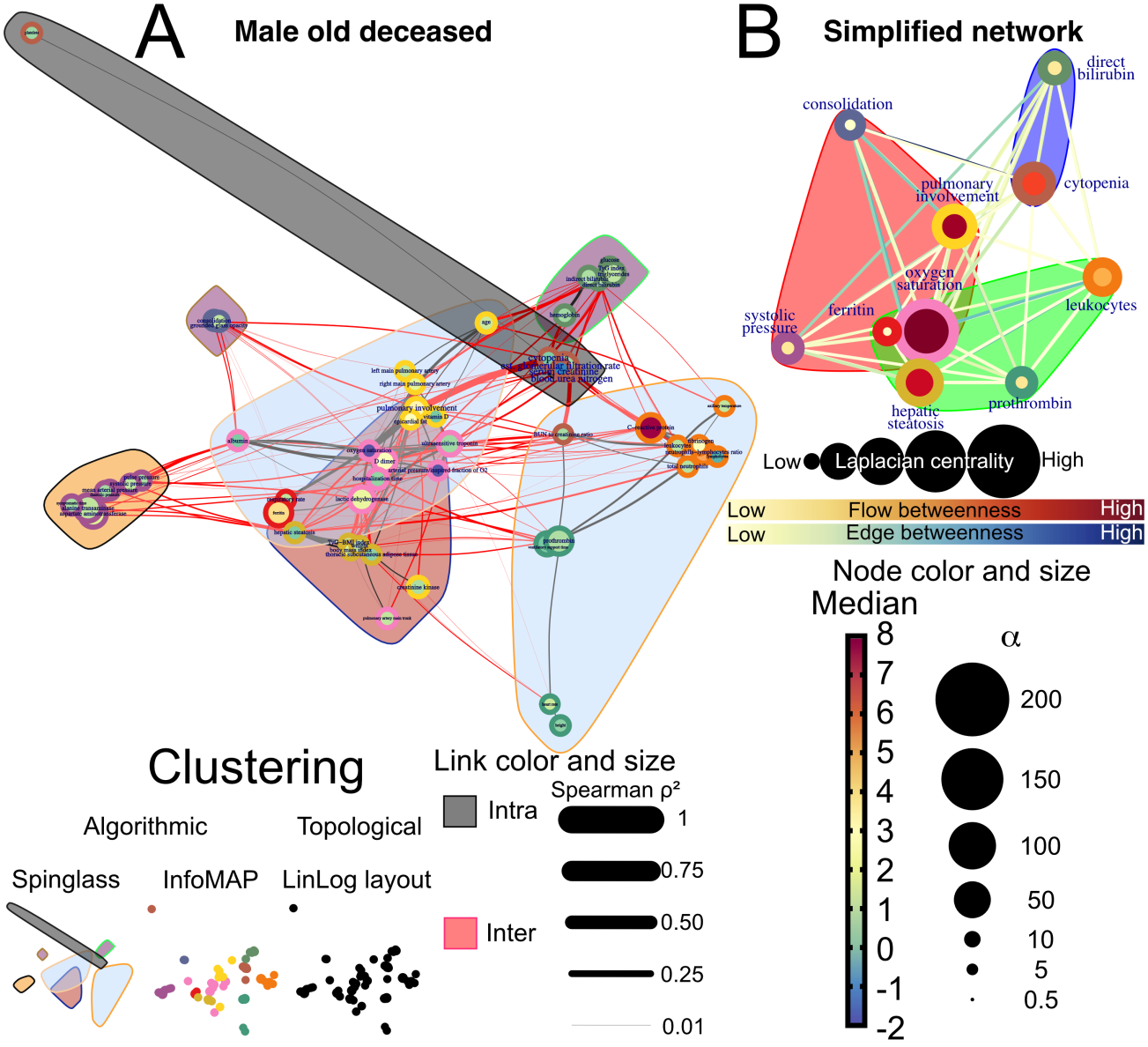
Supplementary Figure 4. Physiological network in male young deceased group. (A) The five study groups' networks were plotted. Links between nodes in the same cluster are shown in black, while shared links are shown in red. Each correlation's strength is indicated by the width of the links. LinLog layout was used to arrange the nodes. The color of the node represents the normalized median value for each study group, and the size represents the deviation from the normal distribution. The color-shaded areas in this network represent spinglass clusters, while the node border represents the nodes that will be collapsed into a single supernode by InfoMAP clustering. (B) depicts a simplified network in which supernodes are labeled with the name of the physiological variable with the greatest influence within the InfoMAP community. Edges were kept, resulting in a network with multiple edges. The color of the edge represents its betweenness. The flow betweenness centrality is represented by node color, and the laplacian centrality is represented by node size.

9 Supplementary Figure 5



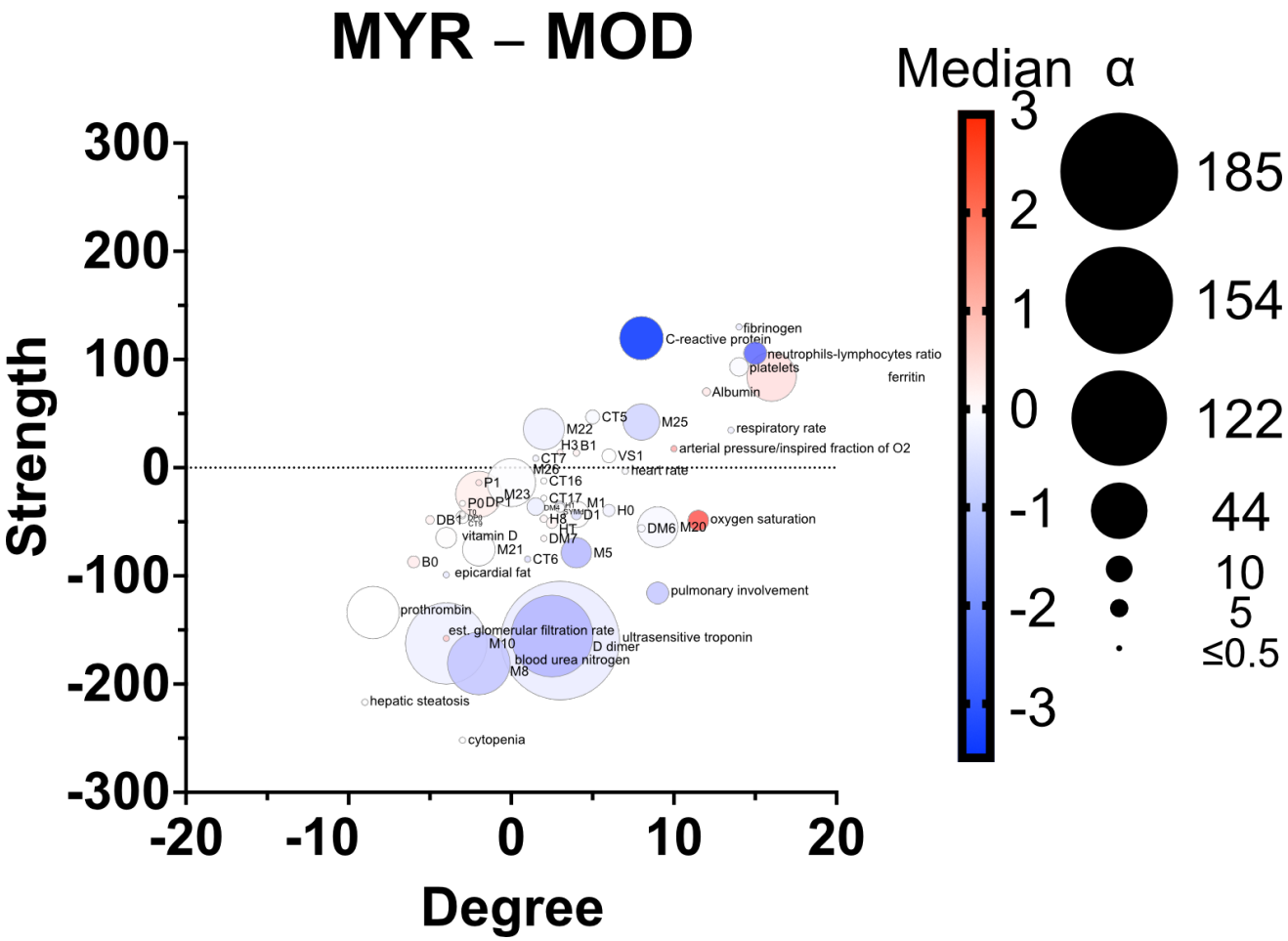
Supplementary Figure 5. Physiological network in male old recovered group. (A) The five study groups' networks were plotted. Links between nodes in the same cluster are shown in black, while shared links are shown in red. Each correlation's strength is indicated by the width of the links. LinLog layout was used to arrange the nodes. The color of the node represents the normalized median value for each study group, and the size represents the deviation from the normal distribution. The color-shaded areas in this network represent spinglass clusters, while the node border represents the nodes that will be collapsed into a single supernode by InfoMAP clustering. (B) depicts a simplified network in which supernodes are labeled with the name of the physiological variable with the greatest influence within the InfoMAP community. Edges were kept, resulting in a network with multiple edges. The color of the edge represents its betweenness. The flow betweenness centrality is represented by node color, and the laplacian centrality is represented by node size.

10 Supplementary Figure 6



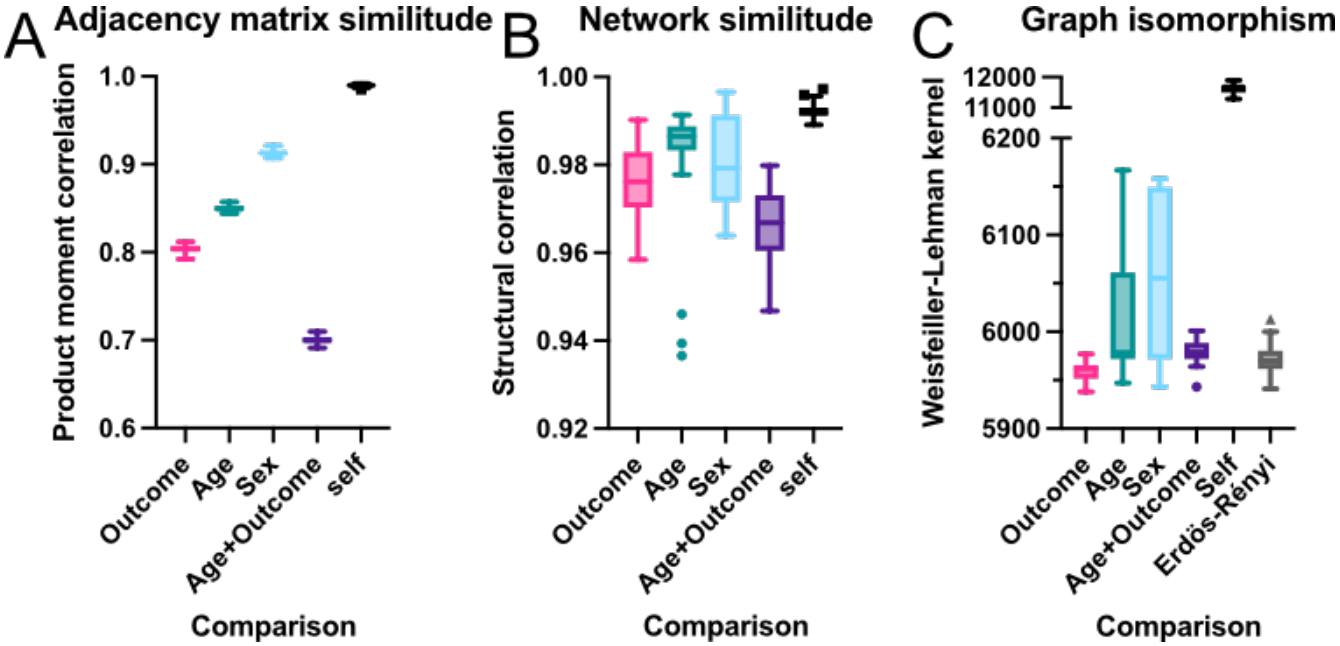
Supplementary Figure 6. Physiological network in male old deceased group. (A) The five study groups' networks were plotted. Links between nodes in the same cluster are shown in black, while shared links are shown in red. Each correlation's strength is indicated by the width of the links. LinLog layout was used to arrange the nodes. The color of the node represents the normalized median value for each study group, and the size represents the deviation from the normal distribution. The color-shaded areas in this network represent spinglass clusters, while the node border represents the nodes that will be collapsed into a single supernode by InfoMAP clustering. (B) depicts a simplified network in which supernodes are labeled with the name of the physiological variable with the greatest influence within the InfoMAP community. Edges were kept, resulting in a network with multiple edges. The color of the edge represents its betweenness. The flow betweenness centrality is represented by node color, and the laplacian centrality is represented by node size.

11 Supplementary Figure 7



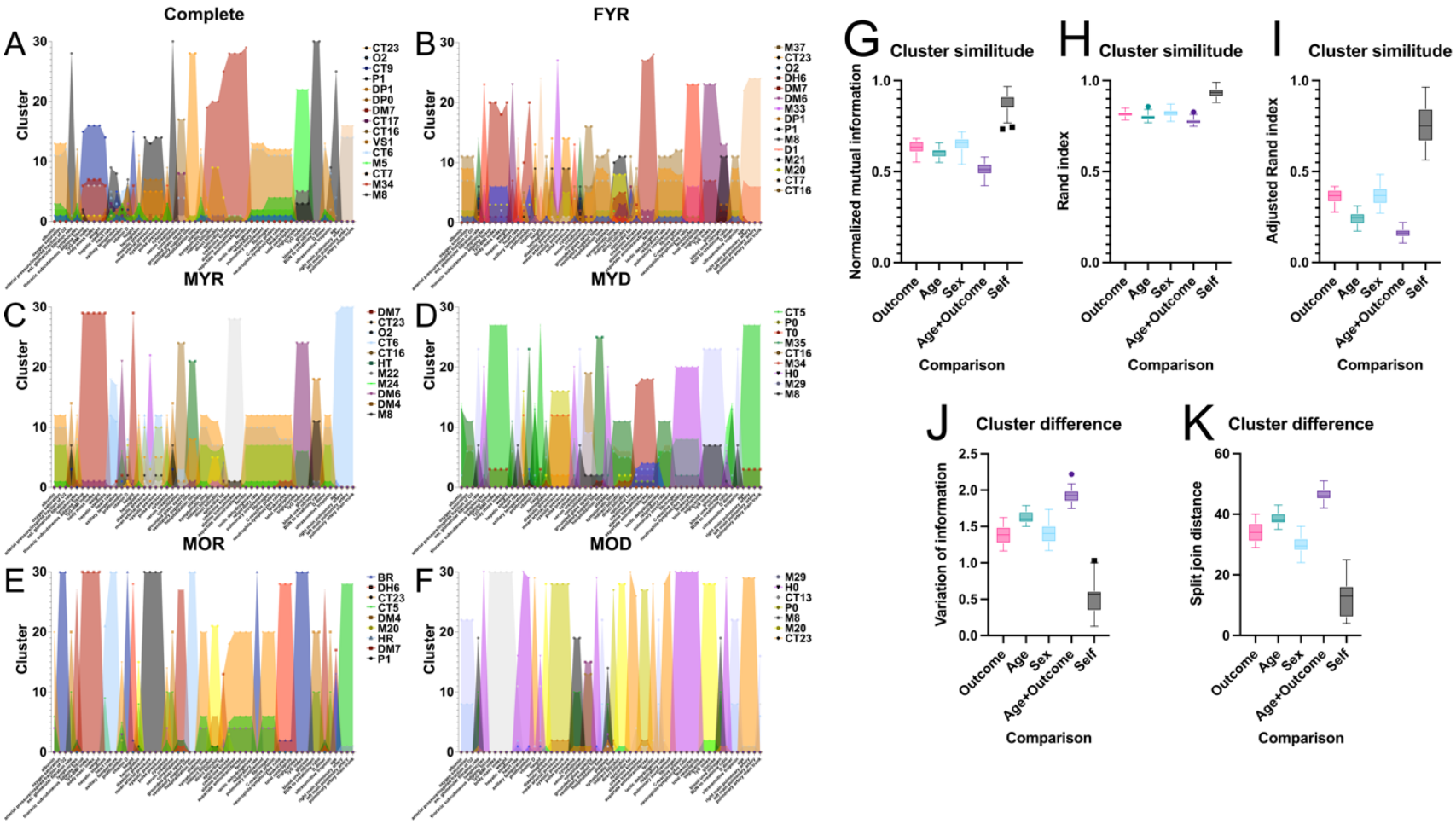
Supplementary Figure 7. Difference between MYR and MOD networks. Physiological variables degree and strength are compared. The gradient of the normalized median value difference is shown in red to blue. The difference between the distributions is indicated by the size of the nodes.

12 Supplementary Figure 8



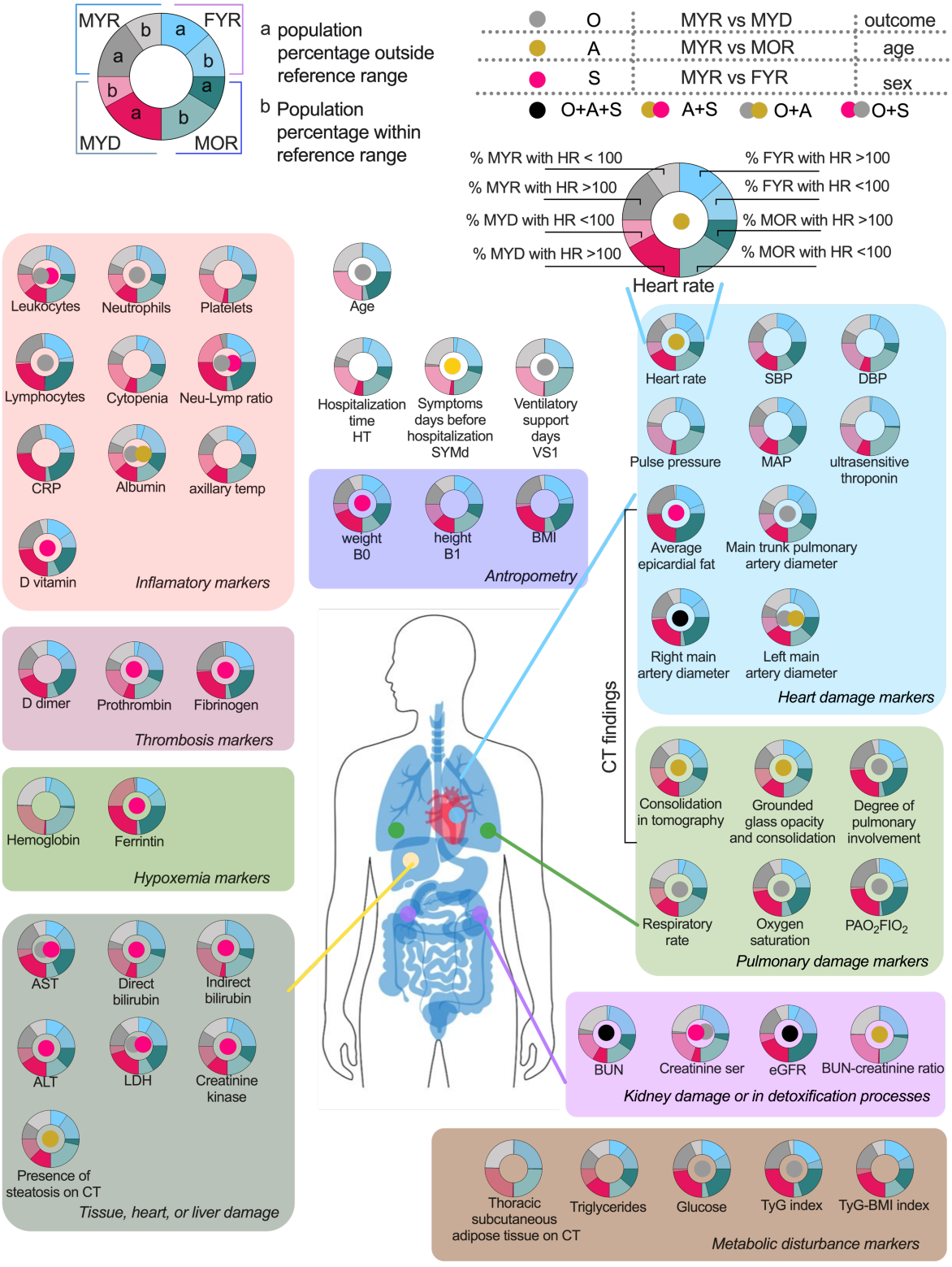
Supplementary Figure 8. Network structure difference. To compare the network structure to the reference, the product-moment correlation (D), structural correlation (F), and Weisfeiler-Lehman isomorphism tests (G) were used. Normalized mutual information (H) was used to quantify cluster similarity, while information variation was used to quantify cluster difference (I). In black we show the comparisson of each network against themselves and in gray against a random Erdös-Rényi network.

13 Supplementary Figure 9



Supplementary Figure 9. Network clusters. Physiological variables were classified into clusters using spinglass algorithm. Clusters were designated after the most relevant variable within each of the 30 network iterations, i.e. the variables with the highest laplacian centrality in each cluster are the core variables. Five clustering comparison methods were employed. In black we show the comparisson of clusters on different iterations of the same network.

14 Supplementary Figure 10



Supplementary Figure 10. Physiological variables differences and pathologic states prevalence among groups. The variables are grouped according to their usefulness as disorders alterations markers. The pie chart graphics show the behavior of the main study groups, the male young recovered (MYR) group is shown in the light blue pair, the female young recovered (FYR) group in the pink pair, the male old recovered (MOR) group in blue and the male young deceased (MYD) group in gray. Each pair is made up of a solid shade (a) and a light shade (b). The solid tone (a) represents the population percentage with values outside the reference limits, while the light tone (b) represents the population percentage with values within the reference limits. The circle in the center indicates the statistically significant differences using Mann-Whitney U. Gray circle indicates a significant difference between the MYR and MYD groups (attributed to the outcome), gold circle indicates the difference between the MYR vs MOR groups (attributed to age), red circle indicates the difference between the MYR vs FYR groups (attributed to sex), while the black circle indicates the presence of statistically significant differences between all groups.