Supplementary (Suppl.) Table 1. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in men with type 1 diabetes; RH 140 (22.5%); no RH 366 (58.9%); Controlled BP 115 (18.5%)

	Model 1	Model 2	Model 3	Model 4	Model 5
DN progression (N=621, 189 events)					
RH	3.34 (2.14, 5.23), <0.0001	3.70 (2.35, 5.83),<0.0001	2.70 (1.67, 4.35), <0.0001	2.21 (1.36, 3.59), 0.001	1.98 (1.21, 3.24), 0.006
No RH	1.06 (0.69, 1.65), 0.8	1.13 (0.73, 1.75), 0.6	1.05 (0.67, 1.66), 0.8	1.05 (0.67, 1.64), 0.8	1.14 (0.72, 1.80), 0.6
Controlled BP	Reference	Reference	Reference	Reference	Reference
Incident CHD (N=564, 132 events)					
RH	2.52 (1.36, 4.67), 0.003	2.03 (1.09, 3.77), 0.02	1.73 (0.89, 3.36), 0.1	1.45 (0.74, 2.83), 0.3	1.41 (0.72, 2.78), 0.2
No RH	1.82 (1.03, 3.22), 0.04	1.49 (0.84, 2.64), 0.2	1.44 (0.79, 2.62), 0.2	1.45 (0.80, 2.65), 0.2	1.46 (0.80, 2.65), 0.3
Controlled BP	Reference	Reference	Reference	Reference	Reference
Incident stroke (N=599, 92 events)					
RH	2.26 (1.17, 4.37), 0.01	2.04 (1.05, 3.95), 0.03	2.00 (0.98, 4.12), 0.06	1.48 (0.70, 3.14), 0.3	1.21 (0.56, 2.62), 0.6
No RH	1.28 (0.70, 2.36), 0.4	1.17 (0.63, 2.15), 0.6	1.30 (0.69, 2.48), 0.4	1.30 (0.69, 2.48), 0.4	1.27 (0.67, 2.41), 0.5
Controlled BP	Reference	Reference	Reference	Reference	Reference
All-cause mortality (N=621, 181 deaths)					
RH	2.16 (1.35, 3.46), 0.001	1.87 (1.17, 2.99), 0.009	1.33 (0.81, 2.21), 0.2	0.95 (0.57, 1.58), 0.8	0.86 (0.51, 1.47), 0.6
No RH	1.24 (0.80, 1.93), 0.3	1.10 (0.71, 1.71), 0.7	1.04 (0.66, 1.63), 0.9	1.00 (0.64, 1.57), 1.0	0.99 (0.63, 1.56), 1.0
Controlled BP	Reference	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years)
Model 3: Model 2 + HbA_{1C}, WHR, triglycerides, smoking and previous CHD and/or previous stroke
Model 4: Model 3 + nephropathy status
Model 5: Model 4 + eGFR/renal stage group (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 2. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in women with type 1 diabetes; RH 66 (13.7%), no RH (56.6%), controlled BP 143 (29.7%)

	Unadjusted	Model 1	Model 2	Model 3	Model 4
DN progression (N=482, 132 events)					
RH					
No RH	(see Suppl. Fig. 2*A)**	(see Suppl. Fig. 2*B*)*	(see Suppl. Fig. 2*C*)*	(see Suppl. Fig. 2*D*)*	(see Suppl. Fig. 2*E*)*
Controlled BP					
Incident CHD (N=451, 107 events)					
RH					
No RH	(see Suppl. Fig. 3*A*)*	(see Suppl. Fig. 3*B*)*	(see Suppl. Fig. 3*C*)*	(see Suppl. Fig. 3*D*)*	(see Suppl. Fig. 3*E*)*
Controlled BP					
Incident stroke (N=465, 46 events)					
RH	7.15 (2.51, 20.31), 0.0002	5.54 (1.91, 16.09),0.002	4.16 (1.37, 12.60), 0.01	3.09 (1.01, 9.46), 0.05	2.44 (0.78, 7.66), 0.1
No RH	3.39 (1.31, 8.75), 0.01	2.63 (0.99, 6.98), 0.05	2.38 (0.89, 6.38), 0.08	2.38 (0.89, 6.36), 0.08	2.34 (0.88, 6.28), 0.09
Controlled BP	Reference	Reference	Reference	Reference	Reference
All-cause mortality (N=482, 121 deaths)					
RH	5.66 (3.17, 10.10), <0.0001	3.97 (2.18, 7.22), <0.0001	2.59 (1.34, 4.99), 0.004	2.26 (1.18, 4.35), 0.01	1.95 (1.02, 3.75), 0.04
No RH	2.35 (1.38, 4.00), 0.002	1.78 (1.02, 3.08), 0.04	1.43 (0.80, 2.55), 0.2	1.46 (0.82, 2.61), 0.2	1.78 (0.99, 3.18), 0.05
Controlled BP	Reference	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group). *Time-varying effect of dependent variable.

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years)
Model 3: Model 2 + HbA_{1C}, WHR, triglycerides, smoking and previous CHD and/or previous stroke
Model 4: Model 3 + nephropathy status
Model 5: Model 4 + eGFR/renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 3. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in individuals with normal AER or microalbuminuria and type 1 diabetes

	Model 1	Model 2	Model 3	Model 4
DN progression (N=630, 95 events)				
RH			0.51 (0.16, 1.61), 0.2	0.50 (0.16, 1.58), 0.2
No RH	(see Suppl. Fig. 4*A*)*	(see Suppl. Fig. 4*B*)*	0.87 (0.51, 1.50), 0.6	0.87 (0.50, 1.49), 0.6
Controlled BP			Reference	Reference
Incident CHD (N=573, 100 events)				
RH	3.74 (1.68, 8.33), 0.001	1.85 (0.81, 4.25), 0.1	1.52 (0.64, 3.63), 0.3	1.51 (0.63, 3.60), 0.3
No RH	2.78 (1.51, 5.11), 0.001	1.84 (0.99, 3.45), 0.05	1.67 (0.88, 3.15), 0.1	1.69 (0.89, 3.19), 0.1
Controlled BP	Reference	Reference	Reference	Reference
Incident stroke (N=616, 56 events)				
RH	4.99 (1.81, 13,78), 0.002	3.19 (1.13, 8.97),0.03	3.55 (1.22, 10.35), 0.02	3.49 (1.20, 10.15), 0.02
No RH	2.60 (1.17, 5.81), 0.02	1.95 (0.86, 4.39), 0.1	2.01 (0.88, 4.58), 0.1	2.01 (0.89, 4.58), 0.09
Controlled BP	Reference	Reference	Reference	Reference
All-cause mortality (N=630, 109 deaths)				
RH	2.93 (1.49, 5.76), 0.002	1.48 (0.74, 2.99), 0.3	1.44 (0.66, 3.10), 0.3	1.32 (0.61, 2.88), 0.5
No RH	1.70 (1.02, 2.81), 0.04	1.15 (0.69, 1.93), 0.6	1.30 (0.77, 2.19), 0.3	1.32 (0.78, 2.22), 0.3
Controlled BP	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group). *Time-varying effect of dependent variable.

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years) and sex
Model 3: Model 2 + HbA_{1C}, WHR/WHR group (men WHR <0.95, 0.96-0.99, ≥1.0; women WHR <0.80, 0.81-0.85, ≥0.86), triglycerides, smoking and previous CHD and/or previous stroke
Model 4: Model 3 + eGFR/renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 4. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in individuals with macroalbuminuria and type 1 diabetes

	Model 1	Model 2	Model 3	Model 4
DN progression (N=473, 226 events)				
RH	2.91 (1.98, 4.28), <0.0001	3.35 (2.24, 5.01), <0.0001	3.18 (2.05, 4.93), <0.0001	2.17 (1.41, 3.34), 0.0004
No RH	0.95 (0.64, 1.41), 0.8	1.02 (0.69, 1.53), 0.9	1.01 (0.66, 1.55), 1.0	0.99 (0.64, 1.51), 0.9
Controlled BP	Reference	Reference	Reference	Reference
Incident CHD (N=442, 139 events)				
RH	1.84 (1.10, 3.07), 0.02	(see Suppl. Fig. 5*A*)*	1.40 (0.80, 2.45), 0.2	1.32 (0.75, 2.33), 0.3
No RH	1.51 (0.93, 2.46), 0.1		1.18 (0.70, 2.00), 0.5	1.18 (0.69, 1.99), 0.5
Controlled BP	Reference		Reference	Reference
Incident stroke (N=450, 82 events)				
RH	2.18 (1.10, 4.33), 0.03	1.62 (0.80, 3.29), 0.2	1.50 (0.69, 3.23), 0.3	1.12 (0.51, 2.45), 0.8
No RH	1.54 (0.79, 3.01), 0.2	1.25 (0.63, 2.47), 0.5	1.25 (0.61, 2.55), 0.5	1.31 (0.64, 2.69), 0.5
Controlled BP	Reference	Reference	Reference	Reference
All-cause mortality (N=473, 193 deaths)				
RH	2.41 (1.51, 3.83), 0.0002	1.83 1.13, 2.95), 0.01	1.32 (0.79, 2.20), 0.3	1.14 (0.68, 1.93), 0.6
No RH	1.71(1.08, 2.70), 0.02	1.32 (0.82, 2.11), 0.2	1.17 (0.72, 1.90), 0.5	1.19 (0.73, 1.93), 0.5
Controlled BP	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group). *Time-varying effect of dependent variable.

Model 1: Unadjusted
Model 2: Adjusted for age and sex
Model 3: Model 2 + HbA_{1C}, WHR/WHR group (men WHR <0.95, 0.96-0.99, ≥1.0; women WHR <0.80, 0.81-0.85, ≥0.86), triglycerides /triglycerides control (triglycerides <2.3, 2.3-4.5, and >4.5 mmol/l), smoking and previous CHD and/or previous stroke
Model 4: Model 3 + eGFR/renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 5. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in individuals with eGFR ≥ 60 ml/min/1.73 m^2 and type 1 diabetes

	Model 1	Model 2	Model 3	Model 4
DN progression (N=749, 111 events)				
RH				
No RH	(see Suppl. Fig. 6*A*)*	(see Suppl. Fig. 6*B*)*	(see Suppl. Fig. 6*C*)*	(see Suppl. Fig. 6*D*)*
Controlled BP				
Incident CHD (N=703, 132 events)				
RH	2.45 (1.20, 5.01), 0.01	1.57 (0.75, 3.27), 0.2	1.40 (0.64, 3.06), 0.4	1.26 (0.57, 2.80), 0.6
No RH	2.48 (1.50, 4.10), 0.0004	1.82 (1.09, 3.06), 0.02	1.77 (1.03, 3.03), 0.04	1.81 (1.05, 3.11), 0.03
Controlled BP	Reference	Reference	Reference	Reference
Incident stroke (N=735, 59 events)				
RH	2.64 (0.91, 7.61), 0.07	1.91 (0.65, 5.62), 0.2	2.30 (0.77, 6.92), 0.1	2.48 (0.82, 7.50), 0.1
No RH	2.46 (1.16, 5.22), 0.02	1.98 (0.92, 4.26), 0.08	2.05 (0.95, 4.44), 0.07	2.16 (1.00, 4.68), 0.05
Controlled BP	Reference	Reference	Reference	Reference
All-cause mortality (N=749, 136 deaths)				
RH	3.25 (1.76, 6.01), 0.0002	2.15 (1.15, 4.02), 0.02	2.20 (1.13, 4.31), 0.02	2.04 (1.04, 4.02), 0.04
No RH	1.91 (1.19, 3.07), 0.007	1.43 (0.88, 2.32), 0.1	1.48 (0.90, 2.45), 0.1	1.50 (0.91, 2.48), 0.1
Controlled BP	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group). *Time-varying effect of dependent variable.

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years) and sex
Model 3: Model 2 + HbA_{1C}/glycemic control (HbA_{1C} <7.5, 7.5-8.99; >9.0 %), WHR, triglycerides, smoking and previous CHD and/or previous stroke
Model 4: Model 3 + eGFR/renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 6. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in individuals with eGFR < 60 ml/min/1.73 m^2 and type 1 diabetes

	Model 1	Model 2	Model 3	Model 4
DN progression (N=349, 210 events)				
RH	1.96 (1.33, 2.89), 0.0007	2.13 (1.43, 3.18), 0.0002	2.25 (1.45, 3.47), 0.0003	2.00 (1.24, 3.22), 0.004
No RH	0.81 (0.54, 1.22), 0.3	0.88 (0.58, 1.33), 0.5	0.95 (0.61, 1.47), 0.8	1.00 (0.62, 1.62), 1.0
Controlled BP	Reference	Reference	Reference	Reference
Incident CHD (N=308, 107 events)				
RH	1.71 (0.95, 3.05), 0.07	1.46 (0.81, 2.63), 0.2	1.31 (0.70, 2.44), 0.4	1.21 (0.64, 2.26), 0.5
No RH	1.38 (0.77, 2.47), 0.3	1.10 (0.61, 1.99), 0.7	0.91 (0.49, 1.71), 0.8	0.87 (0.47, 1.64), 0.7
Controlled BP	Reference	Reference	Reference	Reference
Incident stroke (N=326, 78 events)				
RH	1.87 (0.92, 3.79), 0.08	1.57 (0.77, 3.21),0.2	1.53 (0.72, 3.27), 0.3	1.36 (0.63, 2.96), 0.4
No RH	1.53 (0.75, 3.09), 0.2	1.30 (0.64, 2.63), 0.5	1.43 (0.68, 3.03), 0.3	1.36 (0.64, 2.90), 0.4
Controlled BP	Reference	Reference	Reference	Reference
All-cause mortality (N=349, 165 deaths)				
RH	1.74 (1.07, 2.82), 0.02	1.54 (0.94, 2.52), 0.08	1.16 (0.70, 1.94), 0.5	1.01 (0.60, 1.69), 0.9
No RH	1.48 (0.91, 2.41), 0.1	1.20 (0.73, 1.97), 0.5	0.97 (0.58, 1.60), 0.9	0.90 (0.54, 1.50), 0.7
Controlled BP	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years) and sex
Model 3: Model 2 + HbA_{1C}, WHR, triglycerides/ triglycerides control (triglycerides <2.3, 2.3-4.5, and >4.5 mmol/l), smoking and previous CHD and/or previous stroke
Model 4: Model 3 + eGFR/renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 7. Cox regression models for DN progression, incident CHD and stroke, and all-cause mortality according to the three groups: RH, no RH and controlled BP in individuals with type 1 diabetes when the BP threshold was set <130/80 mmHg

	Model 1	Model 2	Model 3	Model 4	Model 5
DN progression (N=1103, 321 events)					
RH		3.47 (2.41, 5.00), <0.0001	2.76 (1.85, 4.12), <0.0001	2.17 (1.44, 3.26), 0.0002	1.67 (1.11, 2.52), 0.01
No RH	(see Suppl. Fig. 7*A*)*	0.96 (0.68, 1.36), 0.8	0.93 (0.65, 1.34), 0.7	0.91 (0.63, 1.31), 0.6	0.93 (0.65, 1.34), 0.7
Controlled BP		Reference	Reference	Reference	Reference
Incident CHD (N=1015, 239 events)					
RH	2.39 (1.50, 3.80), 0.0002	1.92 (1.20, 3.07), 0.007	1.57 (0.96, 2.59), 0.07	1.43 (0.87, 2.36), 0.1	1.36 (0.82, 2.27), 0.2
No RH	1.45 (0.95, 2.22), 0.08	1.25 (0.81, 1.91), 0.3	1.17 (0.75, 1.82), 0.5	1.22 (0.78, 1.89), 0.4	1.23 (0.79, 1.92), 0.3
Controlled BP	Reference	Reference	Reference	Reference	Reference
Incident stroke (N=1066, 138 events)					
RH	3.16 (1.69, 5.91), 0.003	2.59 (1.38, 4.87), 0.003	2.38 (1.21, 4.68), 0.01	1.90 (0.95, 3.80), 0.07	1.52 (0.75, 3.09), 0.2
No RH	1.59 (0.89, 2.86), 0.1	1.41 (0.78, 2.53), 0.2	1.47 (0.80, 2.72), 0.2	1.53 (0.83, 2.83), 0.2	1.57 (0.85, 2.90), 0.1
Controlled BP	Reference	Reference	Reference	Reference	Reference
All-cause mortality (N=1103, 302 deaths)					
RH	2.96 (1.96, 4,46), <0.0001	2.46 (1.62, 3.72), <0.0001	1.72 (1.11, 2.66), 0.01	1.32 (0.85, 2.06), 0.2	1.23 (0.79, 1.93), 0.3
No RH	1.41 (0.96, 2.08), 0.08	1.25 (0.85, 1.84), 0.3	1.16 (0.79, 1.73), 0.4	1.12 (0.76, 1.66), 0.6	1.13 (0.77, 1.68), 0.5
Controlled BP	Reference	Reference	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group). *Time-varying effect of dependent variable.

Model 1: Unadjusted
Model 2: Adjusted for age/age group (age ≤35, 36-45, 46-55 and >55 years)
Model 3: Model 2 + HbA_{1C}, WHR, triglycerides, smoking and previous CHD and/or previous stroke
Model 4: Model 3 + nephropathy status
Model 5: Model 4 + eGFR/ renal stage (eGFR >90, 60-90 and <60 ml/min/1.73 m^2)

Suppl. Table 8. List of physicians and nurses at each of the FinnDiane centers participating in patient recruitment and characterization

The Finnish Diabetic Nephropathy Study Centers

Anjalankoski Health Center	S.Koivula, T.Uggeldahl
Central Finland Central Hospital, Jyväskylä	T.Forslund, A.Halonen, A.Koistinen, P.Koskiaho, M.Laukkanen, J.Saltevo, M.Tiihonen
Central Hospital of Åland Islands, Mariehamn	M.Forsen, H.Granlund, A.-C.Jonsson, B.Nyroos
Central Hospital of Kanta-Häme, Hämeenlinna	P.Kinnunen, A.Orvola, T.Salonen, A.Vähänen
Central Hospital of Kymenlaakso, Kotka	R.Paldanius, M.Riihelä, L.Ryysy
Central Hospital of Länsi-Pohja, Kemi	H.Laukkanen, P.Nyländen, A.Sademies
Central Ostrobothnian Hospital District, Kokkola	S.Anderson, B.Asplund, U.Byskata, P.Liedes, M.Kuusela, T.Virkkala
City of Espoo Health Center:	
Espoonlahti	A.Nikkola, E.Ritola
Tapiola	M.Niska, H.Saarinen
Samaria	E.Oukko-Ruponen, T.Virtanen
Viherlaakso	A.Lyytinen
City of Helsinki Health Center:	
Puistola	H.Kari, T.Simonen
Suutarila	A.Kaprio, J.Kärkkäinen, B.Rantaeskola
Töölö	P.Kääriäinen, J.Haaga, A-L.Pietiläinen
City of Hyvinkää Health Center	S.Klemetti, T.Nyandoto, E.Rontu, S.Satuli-Autere
City of Vantaa Health Center:	
Korso	R.Toivonen, H.Virtanen
Länsimäki	R.Ahonen, M.Ivaska-Suomela, A.Jauhiainen
Martinlaakso	M.Laine, T.Pellonpää, R.Puranen
Myyrmäki	A.Airas, J.Laakso, K.Rautavaara
Rekola	M.Erola, E.Jatkola
Tikkurila	R.Lönnblad, A.Malm, J.Mäkelä, E.Rautamo
Heinola Health Center	P.Hentunen, J.Lagerstam
Helsinki University Central Hospital, Department of Medicine, Division of Nephrology	M.Feodoroff, D.Gordin, O.Heikkilä, K.Hietala, J.Fagerudd, M.Korolainen, L.Kyllönen, J.Kytö, S.Lindh, K.Pettersson-Fernholm, M.Rosengård-Bärlund, A.Sandelin, L.Thorn, J.Tuomikangas, T.Vesisenaho, J.Wadén
Herttoniemi Hospital, Helsinki	V.Sipilä
Hospital of Lounais-Häme, Forssa	T.Kalliomäki, J.Koskelainen, R.Nikkanen, N.Savolainen, H.Sulonen, E.Valtonen
Hyvinkää Hospital	L. Norvio, A.Hämäläinen
Iisalmi Hospital	E.Toivanen
Jokilaakso Hospital, Jämsä	A.Parta, I.Pirttiniemi
Jorvi Hospital, Helsinki University Central Hospital	S.Aranko, S.Ervasti, R.Kauppinen-Mäkelin, A.Kuusisto, T.Leppälä, K.Nikkilä, L.Pekkonen
Jyväskylä Health Center, Kyllö	K.Nuorva, M.Tiihonen
Kainuu Central Hospital, Kajaani	S.Jokelainen, K.Kananen, M.Karjalainen, P.Kemppainen, A-M.Mankinen, A.Reponen M.Sankari
Kerava Health Center	H.Stuckey, P.Suominen
Kirkkonummi Health Center	A.Lappalainen, M.Liimatainen, J.Santaholma
Kivelä Hospital, Helsinki	A.Aimolahti, E.Huovinen
Koskela Hospital, Helsinki	V.Ilkka, M.Lehtimäki
Kotka Health Center	E.Pälikkö-Kontinen, A.Vanhanen
Kouvola Health Center	E.Koskinen, T.Siitonen
Kuopio University Hospital	E.Huttunen, R.Ikäheimo, P.Karhapää, P.Kekäläinen, M.Laakso, T.Lakka, E.Lampainen, L.Moilanen, S. Tanskanen, L.Niskanen, U.Tuovinen, I.Vauhkonen, E.Voutilainen
Kuusamo Health Center	T.Kääriäinen, E.Isopoussu
Kuusankoski Hospital	E.Kilkki, I.Koskinen, L.Riihelä
Laakso Hospital, Helsinki	T.Meriläinen, P.Poukka, R.Savolainen, N.Uhlenius
Lahti City Hospital	A.Mäkelä, M.Tanner
Lapland Central Hospital, Rovaniemi	L.Hyvärinen, K.Lampela, S.Pöykkö, T.Rompasaari, S.Severinkangas, T.Tulokas
Lappeenranta Health Center	P. Erola, L.Härkönen, P.Linkola, T.Pekkanen, I.Pulli, E.Repo
Lohja Hospital	T.Granlund, K.Hietanen, M.Porrassalmi, M.Saari, T.Salonen, M.Tiikkainen,
Länsi-Uusimaa Hospital, Tammisaari	I.-M.Jousmaa, J.Rinne
Loimaa Health Center	A.Mäkelä, P.Eloranta
Malmi Hospital, Helsinki	H.Lanki, S.Moilanen, M.Tilly-Kiesi
Mikkeli Central Hospital	A.Gynther, R.Manninen, P.Nironen, M.Salminen, T.Vänttinen
Mänttä Regional Hospital	I.Pirttiniemi, A-M.Hänninen
North Karelian Hospital, Joensuu	U-M.Henttula, P.Kekäläinen, M.Pietarinen, A.Rissanen, M.Voutilainen
Nurmijärvi Health Center	A.Burgos, K.Urtamo
Oulaskangas Hospital, Oulainen	E.Jokelainen, P-L.Jylkkä, E.Kaarlela, J.Vuolaspuro
Oulu Health Center	L.Hiltunen, R.Häkkinen, S.Keinänen-Kiukaanniemi
Oulu University Hospital	R.Ikäheimo
Päijät-Häme Central Hospital	H.Haapamäki, A.Helanterä, S.Hämäläinen, V.Ilvesmäki, H.Miettinen
Palokka Health Center	P.Sopanen, L.Welling
Pieksämäki Hospital	V.Sevtsenko, M.Tamminen
Pietarsaari Hospital	M-L.Holmbäck, B.Isomaa, L.Sarelin
Pori City Hospital	P.Ahonen, P.Merisalo, E.Muurinen, K.Sävelä
Porvoo Hospital	M.Kallio, B.Rask, S.Rämö
Raahe Hospital	A.Holma, M.Honkala, A.Tuomivaara, R.Vainionpää
Rauma Hospital	K.Laine, K.Saarinen, T.Salminen
Riihimäki Hospital	P.Aalto, E.Immonen, L.Juurinen

Salo Hospital	A.Alanko, J.Lapinleimu, P.Rautio, M.Virtanen
Satakunta Central Hospital, Pori	M.Asola, M.Juhola, P.Kunelius, M.-L.Lahdenmäki, P.Pääkkönen, M.Rautavirta
Savonlinna Central Hospital	T.Pulli, P.Sallinen, M.Taskinen, E.Tolvanen, T.Tuominen, H.Valtonen, A.Vartia, S-L.Viitanen
Seinäjoki Central Hospital	O.Antila, E.Korpi-Hyövälti, T.Latvala, E.Leijala, T.Leikkari, M.Punkari, N.Rantamäki, H.Vähävuori
South Karelia Central Hospital, Lappeenranta	T.Ensala, E.Hussi, R.Härkönen, U.Nyholm, J.Toivanen
Tampere Health Center	A.Vaden, P.Alarotu, E.Kujansuu, H.Kirkkopelto-Jokinen, M.Helin, S.Gummerus, L.Calonius, T.Niskanen, T.Kaitala, T.Vatanen
Tampere University Hospital	P. Hannula, I.Ala-Houhala, R.Kannisto, T.Kuningas, P.Lampinen, M.Määttä, H.Oksala, T.Oksanen, A.Putila, H.Saha, K.Salonen, H.Tauriainen, S.Tulokas
Tiirismaa Health Center, Hollola	T.Kivelä, L.Petlin, L.Savolainen
Turku Health Center	A.Artukka, I.Hämäläinen, L.Lehtinen, E.Pyysalo, H.Virtamo, M.Viinikkala, M.Vähätalo
Turku University Central Hospital	K.Breitholz, R.Eskola, K.Metsärinne, U.Pietilä, P.Saarinen, R.Tuominen, S.Äyräpää
Vaajakoski Health Center	K.Mäkinen, P.Sopanen
Valkeakoski Regional Hospital	S.Ojanen, E.Valtonen, H.Ylönen, M.Rautiainen,T.Immonen
Vammala Regional Hospital	I.Isomäki, R.Kroneld, L.Mustaniemi, M.Tapiolinna-Mäkelä
Vaasa Central Hospital	S.Bergkulla, U.Hautamäki, V-A.Myllyniemi, I.Rusk

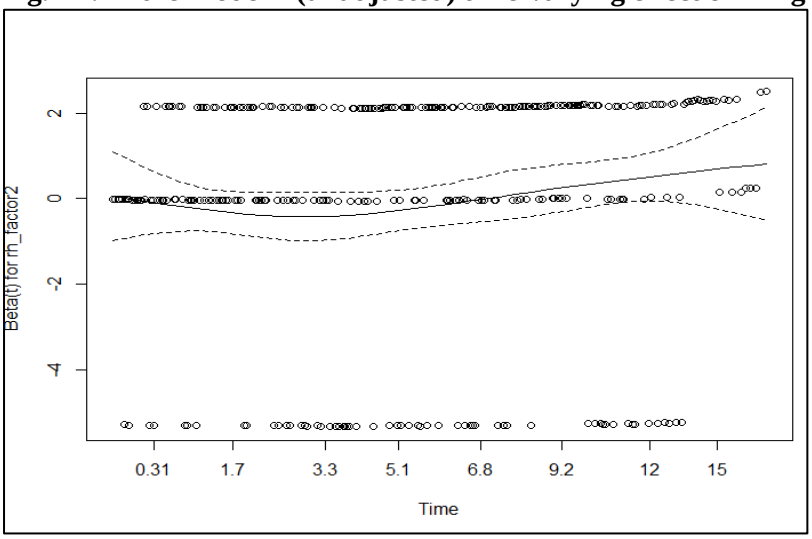
Supplementary figures

To test time-varying effects

The time-dependent effects of the variables were tested by using the Schoenfeld residuals against the follow-up time (*cox.zph*, *Survival package in R*) (30). The assumption is that the hazard rate of an individual is constant over time. When the proportional hazards assumption of the Cox model is not fulfilled, the effect of the covariate is time-varying. When a time-varying effect emerged in an independent variable, we stratified the variable. When the effect occurred in the dependent variable, we visually inspected how the covariate on DN progression (or other outcomes) varied over time (29). Following the method of Zhang et al. (29), we stratified the follow-up time into distinct intervals, so that the proportional hazard assumption was fulfilled for each time interval.

1. Risk of DN progression in all individuals (see Table 1 in the main text)

Fig. 1*A*. In the Model 1 (unadjusted) time-varying effect of RH group 2 (no RH)



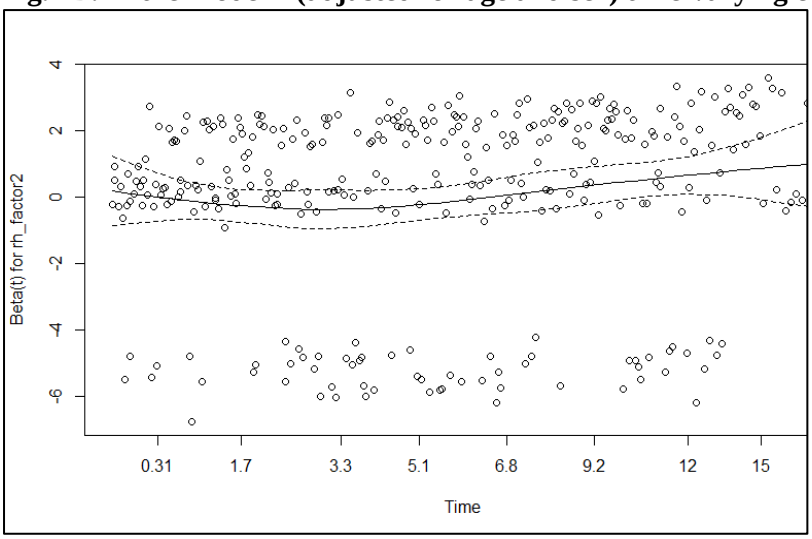
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.0	≥ 5.0
RH	3.49 (2.28, 5.32), <0.0001	3.23 (2.02, 5.15), <0.0001
No RH	0.71 (0.46, 1.11), 0.1	1.26 (0.83, 1.90), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

The risk of DN progression was higher in individuals with RH during the both time periods, while the risk did not differed in those with no RH compared with those who had controlled BP.

Fig. 1*B*. In the Model 2 (adjusted for age and sex) time-varying effect of RH group 2 (no RH)



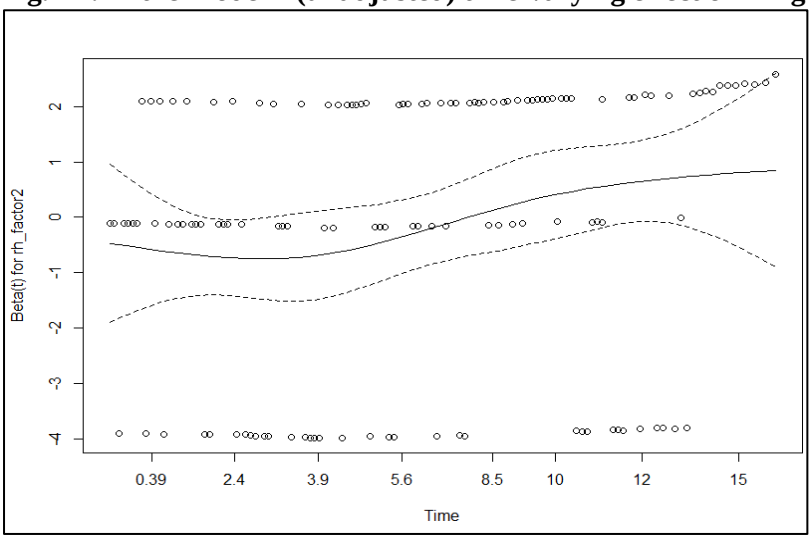
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.5	≥ 5.5
RH	3.63 (2.39, 5.50), <0.0001	4.22 (2.52, 7.04), <0.0001
No RH	0.75 (0.49, 1.14), 0.1	1.53 (0.98, 2.38), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of DN progression was higher in individuals with RH during the both time periods, while the risk did not differed in those with no RH compared with those who had controlled BP.

2. The risk of DN progression in women (see Suppl. Table 2)

Fig. 2*A*. In the Model 1 (unadjusted) time-varying effect of RH group 2 (no RH)

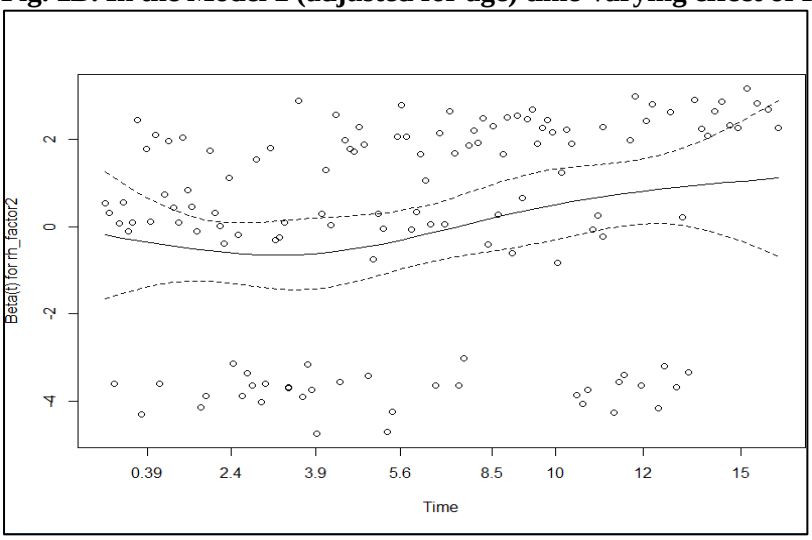


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.4	≥ 4.4
RH	3.19 (1.71, 5.94), 0.0003	4.22 (2.13, 8.38), <0.0001
No RH	0.39 (0.19, 0.79), 0.009	1.43 (0.82, 2.50), 0.2
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of DN progression in those with no RH was even lower during the first time period (< 4.4 years), compared with those who had controlled BP, but no differences were observed afterwards (≥ 4.4 years).

Fig. 2*B*. In the Model 2 (adjusted for age) time-varying effect of RH group 2 (no RH)



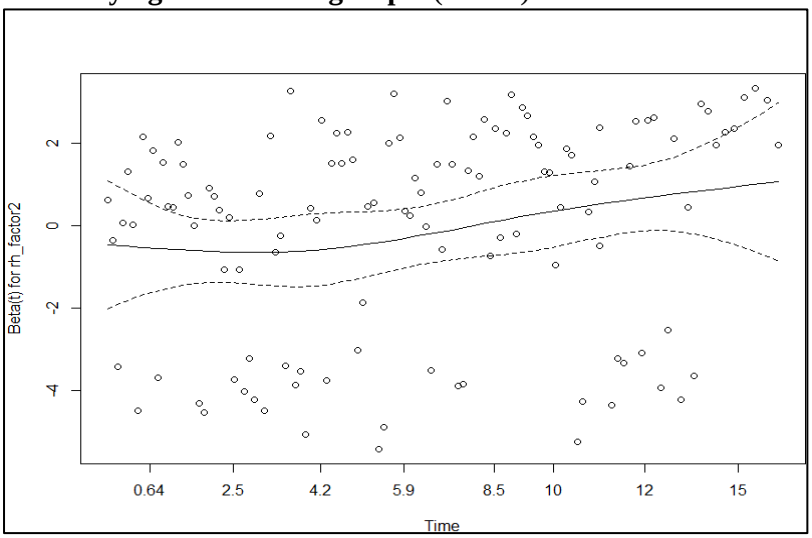
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.0	≥ 4.0
RH	3.67 (1.90, 7.11), 0.0001	5.29 (2.71, 10.34), <0.0001
No RH	0.34 (0.15, 0.75), 0.007	1.63 (0.94, 2.81), 0.08
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

The risk of DN progression in those with no RH was even lower during the first time period (< 4.0 years), compared with those who had controlled BP, but no differences were observed afterwards (≥ 4.0 years).

Fig. 2*C*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous CHD, previous stroke) time-varying effect of RH group 2 (no RH)



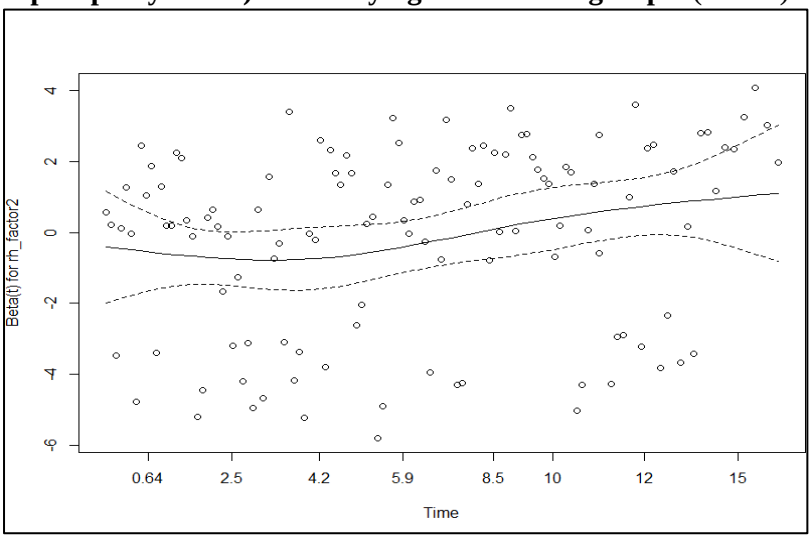
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.0	≥ 4.0
RH	3.14 (1.48, 6.69), 0.003	4.42 (2.15, 9.11), <0.0001
No RH	0.34 (0.14, 0.78), 0.01	1.41 (0.80, 2.49), 0.2
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

The risk of DN progression in those with no RH was even lower during the first time period (< 4.0 years), compared with those who had controlled BP, but no differences were observed afterwards (≥ 4.0 years).

Fig. 2*D*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous CHD, previous stroke, nephropathy status) time-varying effect of RH group 2 (no RH)



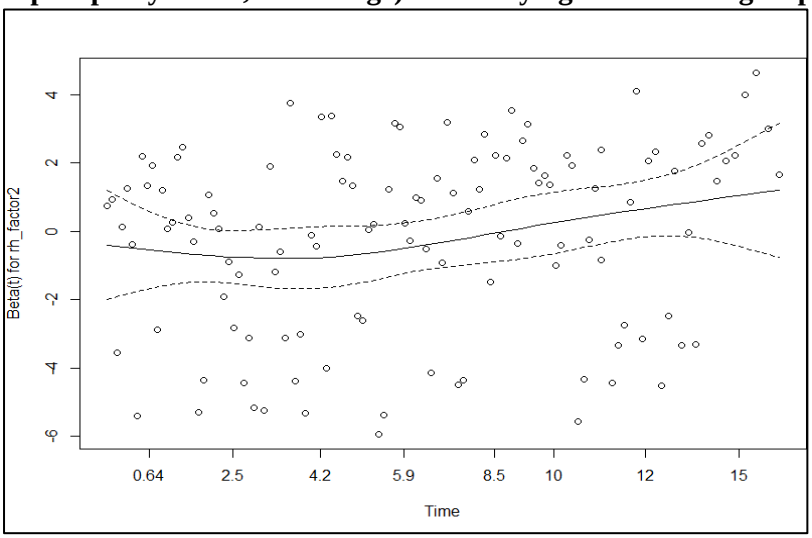
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.2	≥ 4.2
RH	2.47 (1.18, 5.17), 0.01	3.12 (1.49, 6.54), 0.003
No RH	0.32 (0.14, 0.75), 0.009	1.37 (0.77, 2.44), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).

The risk of DN progression in those with no RH was even lower during the first time period (< 4.2 years), compared with those who had controlled BP, but no differences were observed afterwards (≥ 4.2 years).

Fig. 2*E*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous CHD, previous stroke, nephropathy status, renal stage) time-varying effect of RH group 2 (no RH)



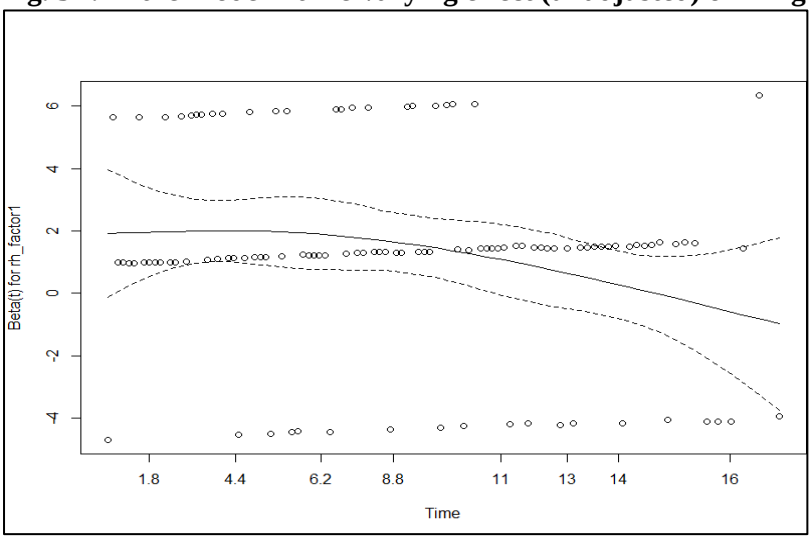
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.2	≥ 4.2
RH	1.78 (0.85, 3.72), 0.1	2.14 (1.01, 4.52), 0.05
No RH	0.33 (0.14, 0.79), 0.01	1.31 (0.73, 2.36), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of DN progression in those with no RH was even lower during the first time period (< 4.2 years), compared with those who had controlled BP, but no differences were observed afterwards (≥ 4.2 years).

3. The risk of CHD in women (see Suppl. Table 2)

Fig. 3*A*. In the Model 1 time-varying effect (unadjusted) of RH group 1 (RH)

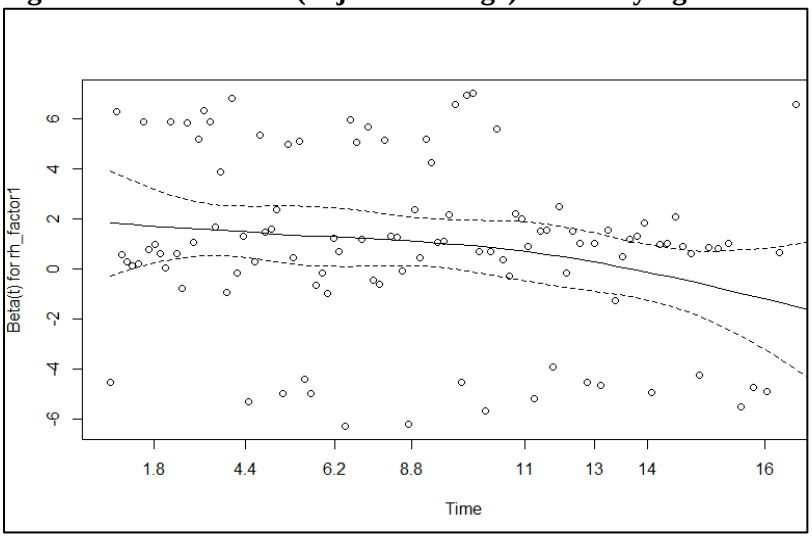


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 6.2	≥ 6.2
RH	5.85 (2.06, 16.60), 0.0009	2.71 (1.23, 5.97), 0.01
No RH	2.65 (1.01, 6.95), 0.05	1.96 (1.07, 3.61), 0.03
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of CHD was higher in individuals with RH during the both time periods (especially during <6.2 years follow-up), and the risk was also higher in those with no RH, compared with those who had controlled BP.

Fig. 3*B*. In the Model 2 (adjusted for age) time-varying effect of RH group 1 (RH)

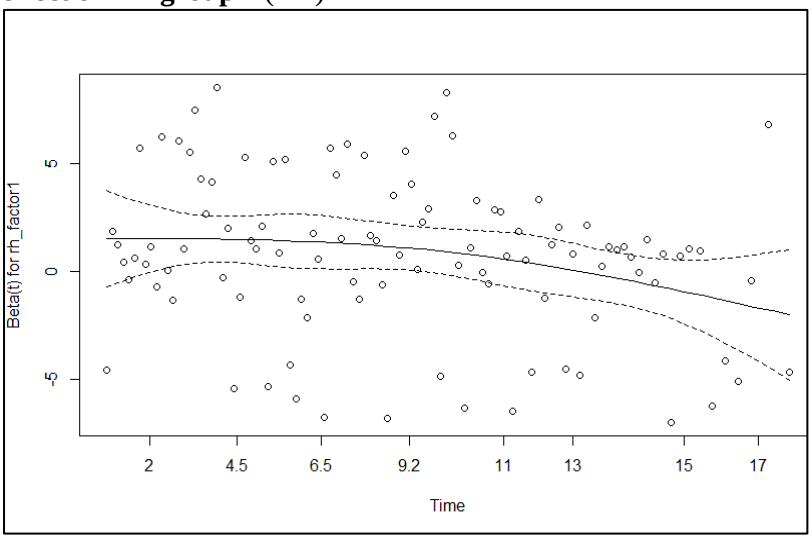


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 6.2	≥ 6.2
RH	3.56 (1.24, 10.26), 0.02	1.76 (0.78, 3.93), 0.2
No RH	1.66 (0.62, 4.42), 0.3	1.38 (0.74, 2.58), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of CHD was higher in individuals with RH only during the first time period (<6.2 years), but not afterwards, while the risk did not differed in those with no RH, compared with those who had controlled BP.

Fig. 3*C*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous stroke) time-varying effect of RH group 1 (RH)

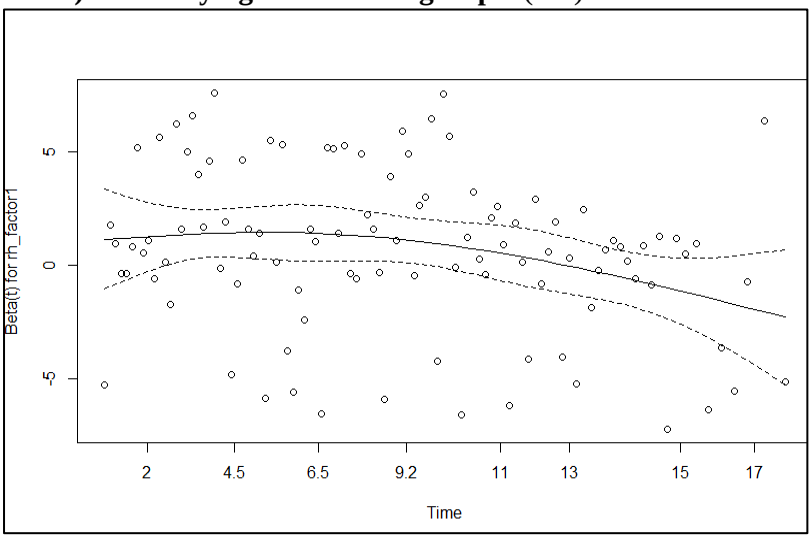


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 6.5	≥ 6.5
RH	2.90 (0.99, 8.51), 0.05	1.65 (0.71, 3.80), 0.2
No RH	1.46 (0.54, 3.93), 0.4	1.38 (0.72, 2.64), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The CHD risk did not differed in those with RH or no RH, compared with those who had controlled BP.

Fig. 3*D*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous stroke, nephropathy status) time-varying effect of RH group 1 (RH)

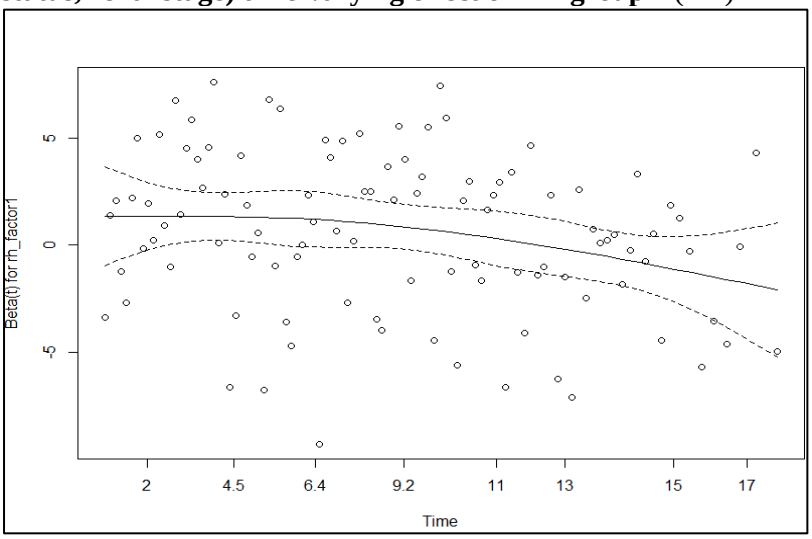


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 9.0	≥ 9.0
RH	2.65 (1.05, 6.65), 0.04	1.15 (0.43, 3.06), 0.8
No RH	1.45 (0.62, 3.38), 0.4	1.39 (0.69, 2.82), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
After full adjustment the CHD risk was higher in women with RH during the first time period (< 9 years), but not afterwards, while the risk did not differed in those with no RH, compared with those who had controlled BP.

Fig. 3*E*. In the Model 3 (adjusted for age, HBA_{1c}, WHR, triglycerides, smoking, previous stroke, nephropathy status, renal stage) time-varying effect of RH group 1 (RH)



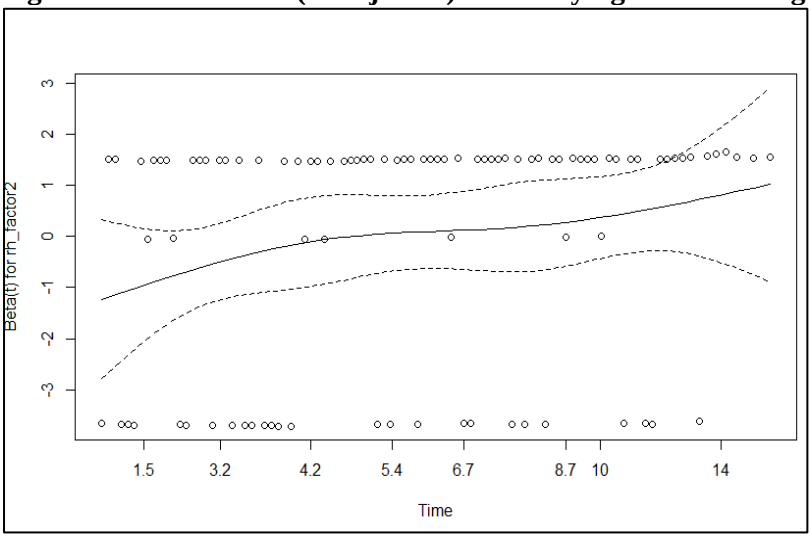
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 9.0	≥ 9.0
RH	2.47 (0.98, 6.23), 0.05	1.09 (0.41, 2.91), 0.8
No RH	1.45 (0.62, 3.37), 0.4	1.40 (0.69, 2.84), 0.3
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The CHD risk did not differed in those with RH or no RH, compared with those who had controlled BP.

4. Risk of DN progression in those with normal AER and microalbuminuria (see Suppl. Table 3)

Fig. 4*A*. In the Model 1 (unadjusted) time-varying effect of RH group 2 (no RH)

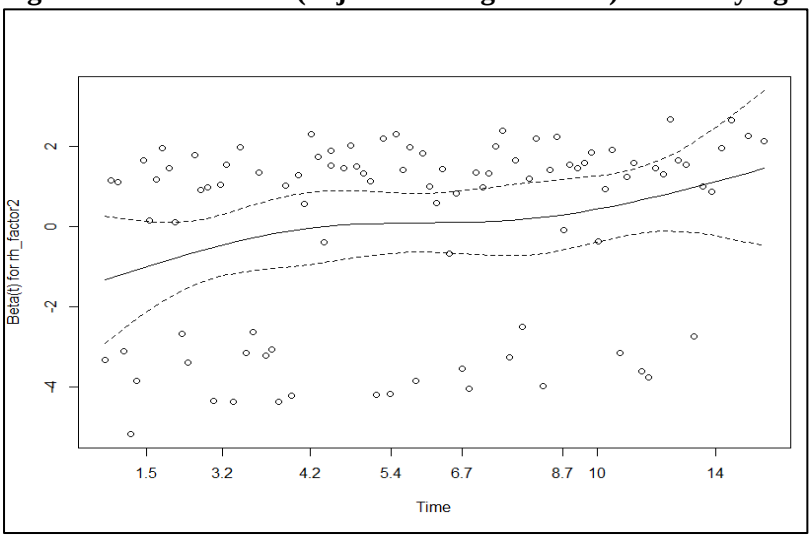


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.4	≥ 5.4
RH	0.81(0.27, 2.41), 0.7	1.08 (0.30, 3.93), 0.9
No RH	0.63 (0.33, 1.18), 0.1	1.55 (0.77, 3.11), 0.2
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The DN progression risk did not differed in those with RH or no RH, compared with those who had controlled BP.

Fig. 4*B*. In the Model 2 (adjusted for age and sex) time-varying effect of RH group 2 (no RH)



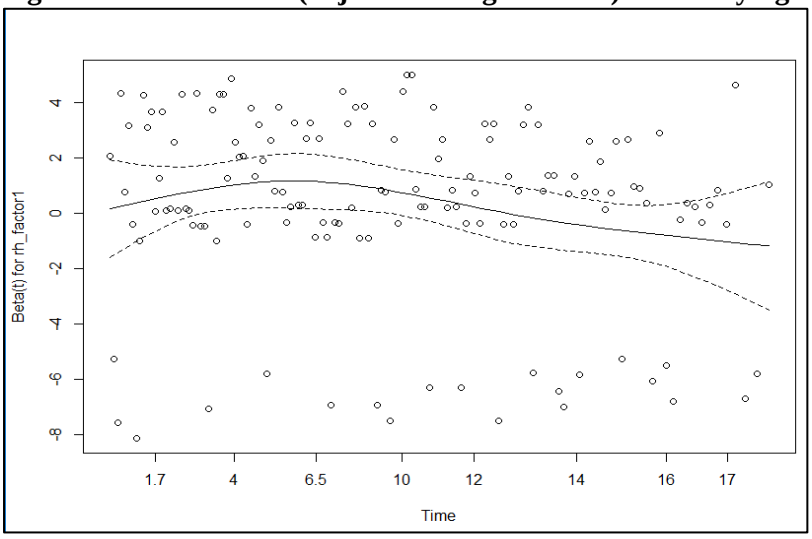
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.2	4.2 – 7.5	> 7.5
RH	0.69 (0.20, 2.39), 0.5	0.94 (0.20, 4.55), 0.9	1.53 (0.30, 7.87), 0.6
No RH	0.43 (0.21, 0.89), 0.02	1.40 (0.60, 3.25), 0.4	1.94 (0.74, 5.09), 0.2
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
During the first 4.2 years the risk of DN progression was slightly lower in those with no RH, while no differences were observed in those with RH, compared with those who had controlled BP.

5. Risk of CHD in individuals with macroalbuminuria (see Suppl. Table 4.)

Fig. 5A. In the Model 2 (adjusted for age and sex) time-varying effect of RH group 1 (RH)



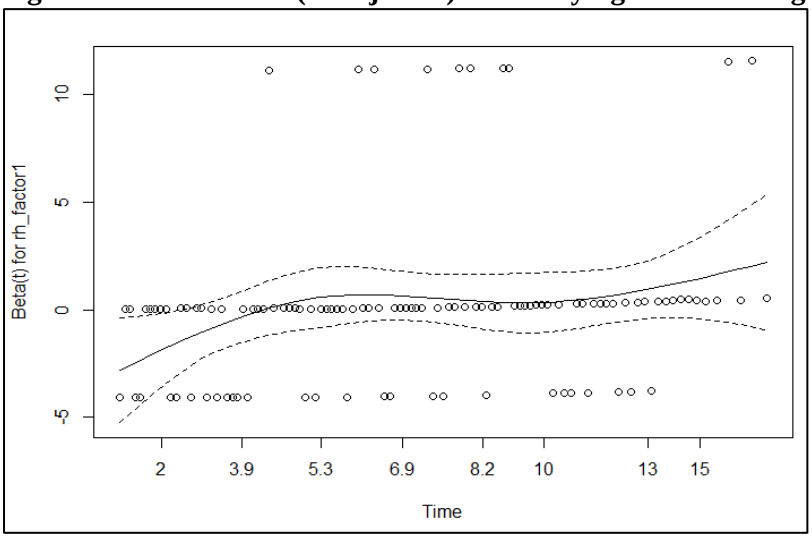
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 4.0	4.0 – 10.0	> 10.0
RH	1.81 (0.59, 5.53), 0.3	2.60 (0.87, 7.77), 0.09	0.89 (0.42, 1.87), 0.8
No RH	1.03 (0.34, 3.12), 1.0	1.60 (0.54, 4.70), 0.4	1.12 (0.59, 2.14), 0.7
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The CHD risk did not differed in those with RH or no RH, compared with those who had controlled BP.

6. Risk of DN progression in those with eGFR ≥60 ml/min/1.73 m^2 (see Suppl. Table 5.)

Fig. 6*A*. In the Model 1 (unadjusted) time-varying effect of RH group 1 (RH)

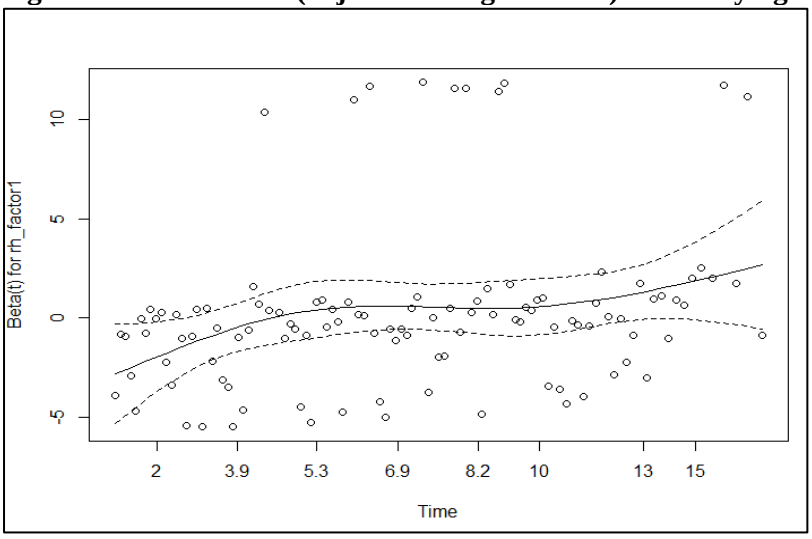


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.3	5.3 – 10.0	> 10.0
RH	0.20 (0.03, 1.56), 0.1	3.54 (1.19, 10.53), 0.02	1.11 (0.23, 5.34), 0.9
No RH	0.70 (0.36, 1.35), 0.3	1.91(0.79, 4.62), 0.1	1.31 (0.56, 3.08), 0.5
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
In individuals with eGFR ≥60 ml/min/1.73 m^2 the risk of DN progression was higher in those with RH during the time-period of 5.3–10.0 years, but no differences were observed between the groups before and after that time period.

Fig. 6*B*. In the Model 2 (adjusted for age and sex) time-varying effect of RH group 1 (RH)

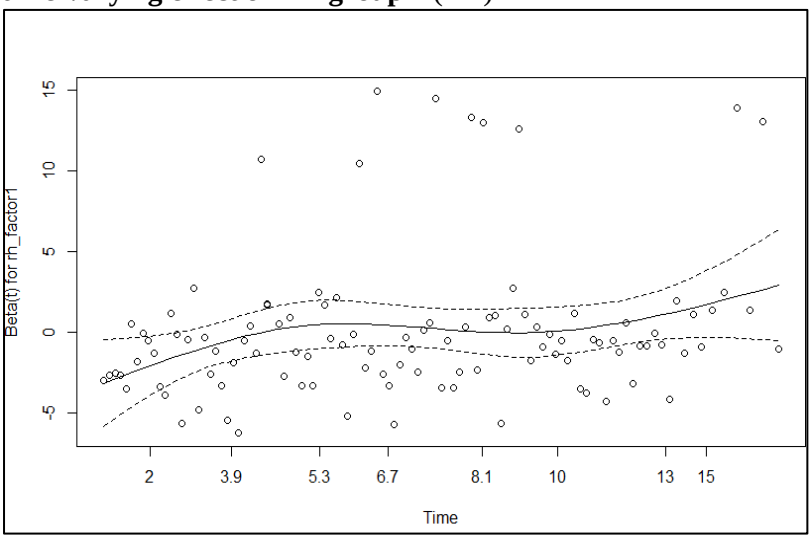


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.3	5.3 – 10.0	> 10.0
RH	1.00 (0.33, 3.08), 1.0	1.09 (0.22, 5.31), 0.9	1.08 (0.12, 9.74), 0.9
No RH	0.71 (0.37, 1.36), 0.3	1.38 (0.58, 3.24), 0.5	1.65 (0.54, 5.00), 0.4
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The DN progression risk did not differed in those with RH or no RH, compared with those who had controlled BP during the follow-up.

Fig. 6*C*. In the Model 3 (adjusted for age, sex, HBA_{1c}, waist, triglycerides, smoking, previous CHD and stroke) time-varying effect of RH group 1 (RH)

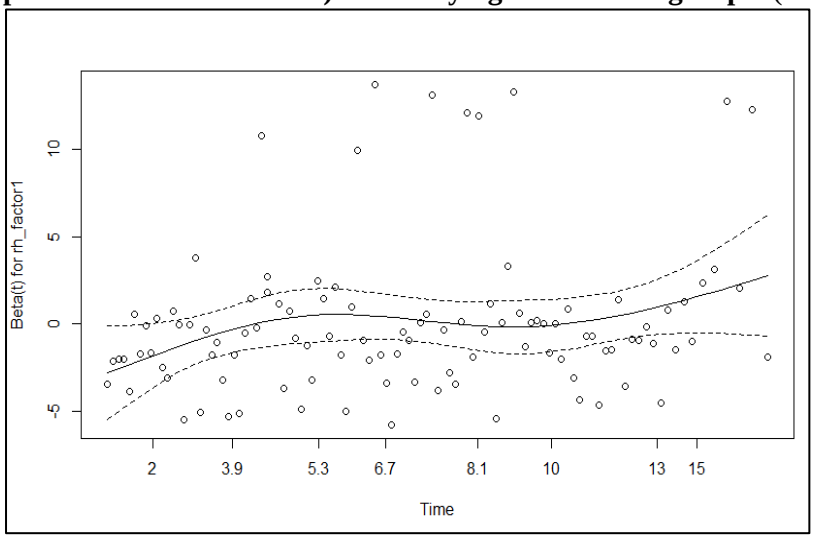


By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.5	5.5 – 10.0	> 10.0
RH	0.19 (0.02, 1.47), 0.1	2.49 (0.78, 7.91), 0.1	0.89 (0.18, 4.38), 0.9
No RH	0.67 (0.34, 1.32), 0.2	1.71(0.70, 4.17), 0.2	0.98 (0.41, 2.37), 1.0
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The DN progression risk did not differed in those with RH or no RH, compared with those who had controlled BP during the follow-up.

Fig. 6*D*. In the Model 4 (adjusted for age, sex, HBA_{1c}, waist, triglycerides, smoking, nephropathy status, previous CHD and stroke) time-varying effect of RH group 1 (RH)



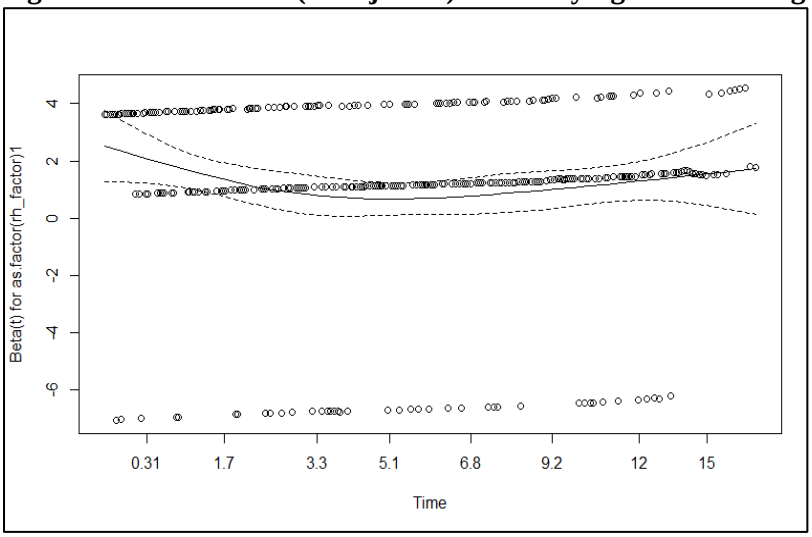
By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.5	5.5 – 10.0	> 10.0
RH	0.19 (0.02, 1.52), 0.1	2.54 (0.80, 8.10), 0.1	0.91 (0.18, 4.46), 0.9
No RH	0.66 (0.34, 1.32), 0.2	1.71 (0.70, 4.16), 0.2	0.98 (0.40, 2.36), 1.0
Controlled BP	Reference	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The DN progression risk did not differed in those with RH or no RH, compared with those who had controlled BP during the follow-up.

7. The Risk of DN progression in all individuals with the BP threshold of < 130/80 mmHg (Suppl. Table 7.)

Fig. 7*A*. In the Model 1 (unadjusted) time-varying effect of RH group 1 (RH)



By splitting the follow-up, the proportional hazard assumption was fulfilled in each time-interval.

Follow-up (years)	< 5.1	≥ 5.1
RH	3.44 (2.09, 5.65), <0.0001	2.91 (1.73, 4.89), <0.0001
No RH	0.75 (0.46, 1.24), 0.3	1.11 (0.69, 1.77), 0.7
Controlled BP	Reference	Reference

Data are HR (95% CI) and p-values (Controlled BP reference group).
The risk of DN progression was higher in individuals with RH during the both time periods, while the risk did not differed in those with no RH compared with those who had controlled BP.