**SUPPLEMENTARY FIGURE LEGENDS**

**Supplementary Figure 1.** Soluble ACE2 (sACE2) activity in sera from (A) 1208 and (B) 1192 participants 3 months after SARS-CoV-2 exposure stratified by chronic precondition. The corresponding ICD-10 codes for each disease group are shown in parentheses and diseases affecting <10 participants were combined into the group any other chronic disease. Participants with diabetes mellitus (n=14) or chronic kidney disease (CKD, n=2) had higher sACE2 activity than healthy participants and were therefore excluded in (B). Mean and 1 ± times standard deviations are shown in black and individual data for each participant are depicted in gray.  *p* values were calculated using a one-way ANOVA (A: F[11, 1570] = 3.31, *p* = 0.0002; B: F[10, 1528] = 1.13, *p* = 0.33) with Dunnett’s correction for comparing participants with no chronic disease (group 1) with each disease group. Only p-values < 0.1 are shown.

**Supplementary Figure 2.** Distribution of study participants classified as exposed (light red; n=598) or infected (light green; n=594) based on RT-PCR and/or serology (seropositive: positive in at least 2 assays; seronegative: negative in at least 2 assays)

**Supplementary Figure 3.** (A, C, E)Distribution of anti-SARS-CoV-2 antibody responses in sera from 773 participants without (w/o) positive RT-PCR and (B, D, F) frequency of anti-SARS-CoV-2 levels measured in 598 participants classified as seronegative (non-infected). Antibody responses were determined against (A, B) anti-N pan-Ig, (C, D) anti-S1-RBD IgG and (E, F) anti-S1 IgG. Dashed lines indicate cutoffs for positivity as per the manufacturer’s instructions.

**Supplementary Figure 4.** Soluble ACE2 (sACE2) activity in sera from unexposed pre-pandemic controls (*n* = 154) and participants 3 months after SARS-CoV-2 household exposure with and without evidence of infection based on different definitions of seropositivity. Participants were classified as infected with a positive RT-PCR test result or seropositive in (A) anti-N pan-Ig, anti-S1-RBD IgG or anti-S1 IgG, (B) only anti-N pan-Ig, (C) only anti-S1-RBD IgG or (D) only anti-S1 IgG. Mean and 1 ± times standard deviation are shown in black. *p* values were calculated using one-way ANOVA (A: F[2, 1343] = 61.8 *p*<0.0001; B: F[2, 1342] = 61.8, *p*<0.0001; C F[2, 1341] = 61.8, *p*<0.0001; D F[2, 1339] = 61.9, *p*<0.0001;) with Tukey correction for multiple comparisons.

**Supplementary Figure 5.** Longitudinal soluble ACE2 (sACE2) activity from (A) 263 participants from Tübingen and (B) 82 participants from Heidelberg 3 to 12 months after SARS-CoV-2 exposure. In (c) difference of sACE2 (∆sACE2) activity in sera from 345 participants 3 (T1) to 12 (T2) months after SARS-CoV-2 exposure was stratified by study site. Individual data for each participant are depicted in gray. (A, B) longitudinal mean and (B) mean and 1 ± times standard deviation are shown in black. *p* value was calculated using (A, B) paired and (C) unpaired t-test.

**Supplementary Figure 6.** Difference of soluble ACE2 (∆sACE2) activity in sera from 345 participants 3 months (T1) to 1 year (T2) after SARS-CoV-2 exposure stratified by age group. Mean and 1 ± times standard deviations are shown in black and individual data for each participant are depicted in gray.  *p* values were calculated using unpaired t-test.

**Supplementary Figure 7.** Soluble ACE2 (sACE2) activity in sera from (A) 361 participants 3 months and (B) 236 participants 1 year after SARS-CoV-2 infection stratified by moderate/severe persistent symptoms still present 1 year after SARS-CoV-2. Mean and 1 ± times standard deviation are shown in black and individual data for each participant are depicted in gray.  *p* values were calculated using unpaired t-test.