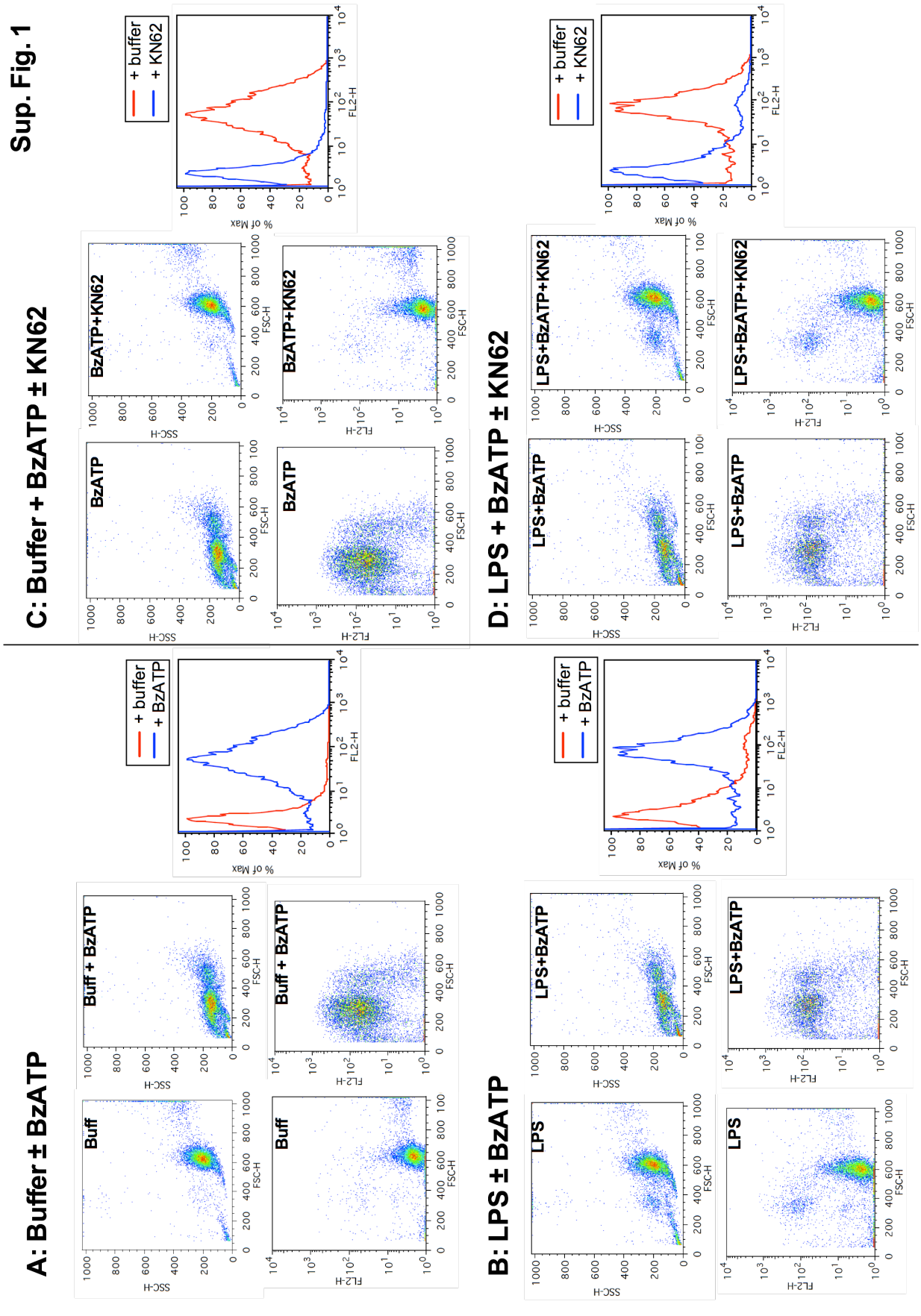
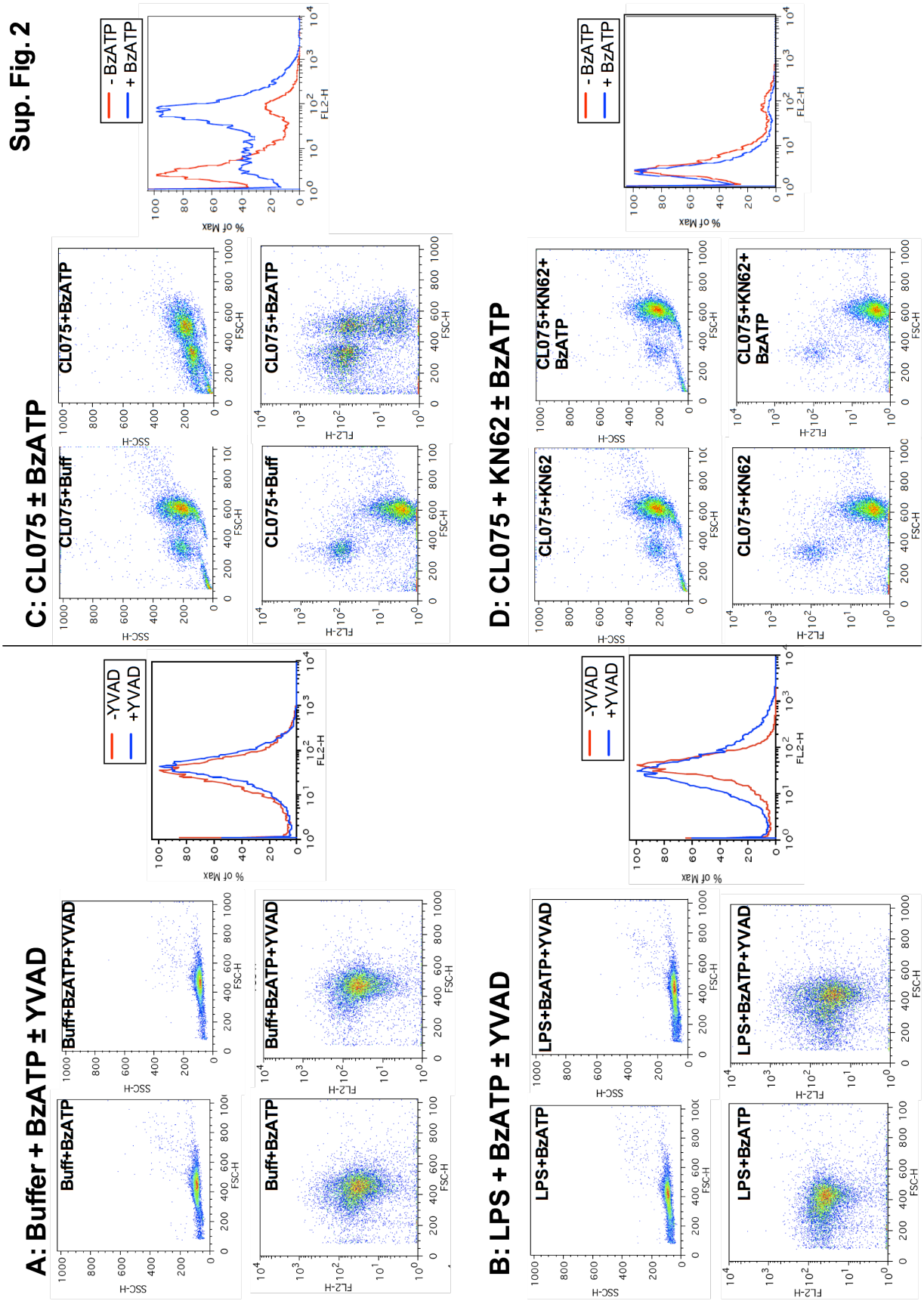
Supplemental Figure 1: Annexin V binding to monocytes, measured by flow cytometry, is dependent upon P2X7 receptor activation but independent of IL-1β release. PS exposure was assessed by flow cytometry for monocytes labelled with annexin V-PE, under the conditions shown. For each treatment forward-scatter against side-scatter dot plots are shown for all flow cytometric events (ungated) in upper panels, and corresponding forward-scatter against FL2 fluorescence channel dot plots are shown for all events (ungated) in lower panels. The corresponding histograms of annexin V staining for both treatments are shown centre-right. *A*, PS staining due to BzATP: monocytes were primed with buffer for 3 h followed by buffer or BzATP for 20 min or *B*, with LPS (1 ng/ml) for 3 h then stimulated with buffer or BzATP for 20 min. *C*, PS exposure inhibition by a P2X7 receptor antagonist: monocytes primed with buffer (control), *C*, or LPS (1 ng/ml)*, D,* for 3 h in the presence of DMSO (vehicle control) or KN62, then stimulated with Buffer or BzATP for 20 min. The data shown is from a single donor (different from that shown in Fig. 6), representative of a total of 4 separate experiments from separate donors.

Supplemental Figure 2: Annexin V binding to monocytes, measured by flow cytometry, is dependent upon P2X7 receptor activation but independent of IL-1β processing and release. PS exposure was assessed by flow cytometry for monocytes labelled with annexin V-PE, under the conditions shown. For each treatment forward-scatter against side-scatter dot plots are shown for all flow cytometric events (ungated) in upper panels, and corresponding forward-scatter against FL2 fluorescence channel dot plots are shown for all events (ungated) in lower panels. The corresponding histograms of annexin V staining for both treatments are shown centre-right. The effect of caspase-1 inhibition on PS exposure: monocytes primed with buffer (control), *A*, or LPS (1 ng/ml), *B*, for 3 h in the presence of DMSO (vehicle control) or YVAD caspase-1 inhibitor, then stimulated with Buffer or BzATP for 20 min. The effect of CL075 on PS exposure at 3 h in the presence of DMSO (vehicle control), *D*, or KN62, *C*. Monocytes treated with CL075 were subsequently stimulated with buffer or BzATP for 20 minutes in the absence or presence of KN62 The data shown are from a single donor for treatments in parts A and B, representative of a total of 3 separate experiments from separate donors. Data shown in parts C and D are from another single donor, representative of a total of 4 separate experiments from separate donors (each different from that shown in Fig. 6).

Sup. Fig. 1


A: Buffer ± BzATP
Buff
Buff + BzATP
+ buffer
+ BzATP
% of Max
B: LPS ± BzATP
LPS
LPS+BzATP
C: Buffer + BzATP ± KN62
BzATP
BzATP+KN62
+ buffer
+ KN62
D: LPS + BzATP ± KN62
LPS+BzATP
LPS+BzATP+KN62
SSC-H
FSC-H
FL2-H


Sup. Fig. 2
A: Buffer + BzATP ± YVAD
Buff+BzATP
Buff+BzATP+YVAD
-YVAD
+YVAD
B: LPS + BzATP ± YVAD
LPS+BzATP
LPS+BzATP+YVAD
C: CL075 ± BzATP
CL075+Buff
CL075+BzATP
- BzATP
+ BzATP
D: CL075 + KN62 ± BzATP
CL075+KN62
CL075+KN62+ BzATP
SSC-H
FSC-H
FL2-H
% of Max