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Apparent Joint Swarms Formed by the Crack-Jump Process

D. C. P. Peacock[1] | B. Leiss[1] | M. W. Anderson[2,3]

[1]University of Göttingen, Göttingen, Germany | [2]School of Geography, Earth and Environmental Sciences, University of Plymouth, Plymouth, UK | [3]School of Environmental Sciences, University of Hull, Hull, UK

Correspondence: B. Leiss (bleiss1@gwdg.de)

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ABSTRACT

Joint swarms can be important components of fractured reservoirs. They are often explained as damage around faults or related to mechanical differences between layers, although this does not explain the close spacing of the joints. Joint swarms around Bergen (Norway) are described, which are not related to exposed faults and are not influenced by layering or foliation in the Lower Palaeozoic gneisses. We suggest an evolution whereby: (1) a zone of microcracks develops; (2) one microcrack propagates and becomes connected to a source of mineralising fluid; (3) the fracture becomes a microvein, with a higher tensile strength than the microcracked host rock; (4) another microcrack propagates and the cycle is repeated, producing a zone of microveins; (5) the veins are partly weathered out, producing an apparent joint swarm, or the microveins crack at or near the ground-surface. Joint swarms in exposed analogues may therefore not occur at reservoir depths.

1 | Introduction

A *fracture corridor* is a tabular zone of significantly increased fracture intensity (e.g., Questiaux, Couples, and Ruby 2010). Gabrielsen and Braathen (2014) suggest a fracture corridor that is dominated by joints should be termed *joint swarm*. The examples described here show joints with millimetre- to centimetre-scale spacings but that are exposed for heights of several metres. They are therefore more closely spaced than predicted by models that suggest new joints will not form in the stress shadows around existing joints (e.g., Becker and Gross 1996; Storti et al. 2022). Relationships between layer thickness and joint spacing are described in terms of a maximum joint frequency, or *saturation* (e.g., Gross et al. 1995; Tan et al. 2014). Although fracture corridors are commonly explained in terms of damage around faults (e.g., Souque et al. 2019; de Joussineau 2023), the fracture corridors described here show no evidence for faulting. Besides, a damage zone model would not explain why the joints in a joint swarm are *oversaturated* (Underwood et al. 2003). Fracture corridors have also been explained in terms of contrasts in the mechanical properties of layers (e.g., de Joussineau and Petit 2021). Layering does not appear to influence joints in the gneisses that host the fracture corridors described here.

The aims of this paper are as follows: (1) to describe selected fracture corridors (apparent joint swarms) in the gneisses around Bergen, Norway; (2) to suggest a model to explain the observation that the joints appear to be related to microveins (sensu Caputo and Hancock 1999); (3) to suggest other potential origins; and (4) to discuss the implications for making predictions about fracturing of reservoir rocks.

2 | Geological Background

This paper describes fracture corridors in the gneisses around Bergen, Norway (Figure 1). The area shows metasedimentary and igneous rocks that were deformed during the Caledonian Orogeny (e.g., Fossen 1989, 1998; Putnis, Jamtveit, and Austrheim 2017). Various generations of post-Caledonian fractures occur

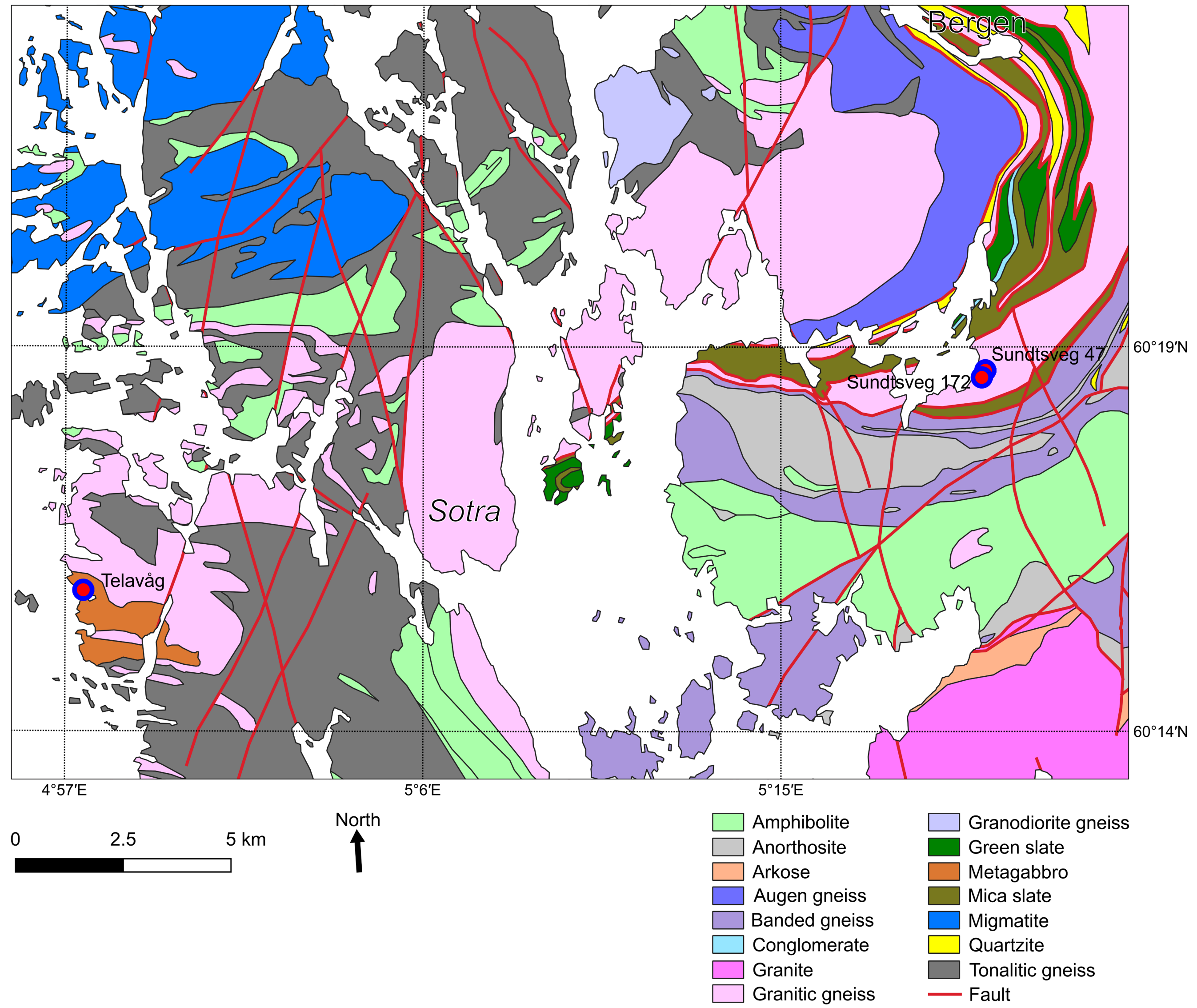

FIGURE 1 | Map of the Bergen area showing the locations of the field examples. Geology is from the NGU 1:250,000 scale sheets (downloaded from https://www.ngu.no/geologiske-kart on 17 December 2023).

(Fossen 1998; Larsen et al. 2003). Hermansen (2019) gives detailed descriptions of apparent joint swarms, while Sanderson and Peacock (2019) use examples from the area to illustrate the measurement and analysis of fracture systems. Moore et al. (2020) describe microveins in the region.

The main location analysed is an excavation behind a block of apartments at Sundtsveg 172, Nesttun, Vestland (60°18′49"N, 5°20′03″ E), but similar structures have also been observed in a road-cut at Sundtsveg 47, Nesttun (60°18′53"N, 5°20′07″ E), ~200 m to the NE. Both of these locations are mapped by the Norges Geologiske Undersøkelse (NGU) as 'gneiss, mostly granitic migmatite, augen and banded gneiss', within the Ulriken Gneiss Complex (Fossen 1989). Similar structures also occur in coastal exposures near Telavåg, Øygarden (60°16′39"N, 4°57′36″ E; Hermansen 2019). The Telavåg location is within the Precambrian Øygarden Complex (e.g., Fossen 1998), and shows quartzo-feldspathic gneisses, although it is mapped by the NGU as being in metagabbro (Figure 1).

3 | Veins, Joints and Fracture Corridors at Three Locations

Apparent joint swarms that appear to be formed from the weathering out of microveins have been analysed (Figure 1), none of which show evidence for faulting.

3.1 | Sundtsveg 172

This ~0.51-m wide apparent joint swarm is in gneiss with a foliation that dips ~40° towards 130°, and consists of at least 10 joints that dip ~72° towards 344° (Figure 2). The joints show coatings of a dark green material and are parallel to and follow microveins that are <2 mm wide. Zones of pale alteration occur ~10 mm on either side of some of the microveins. Some microveins do not have joints, with an example having been examined using a scanning electron microscope (Figure 3). The mineral in the microveins has been identified as chlorite. The

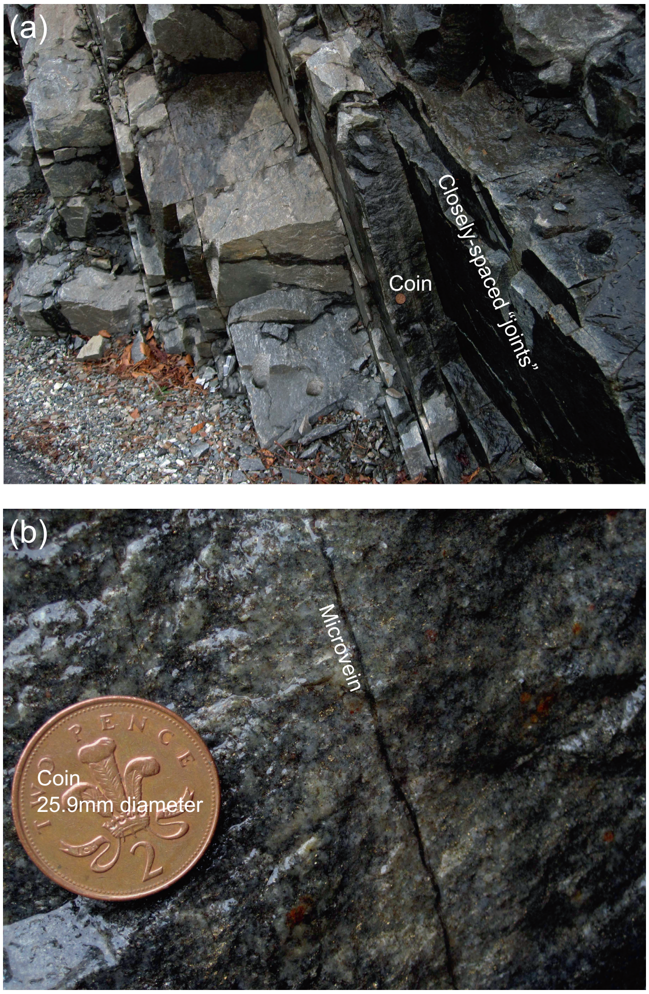

FIGURE 2 | Photographs of the fracture corridor at Sundtsveg 172, Nesttun, near Bergen, Norway (60°18′53.56″N, 5°20′6.80″E). (a) Closely spaced joints in Lower Palaeozoic metamorphic rocks. (b) Close-up of a chlorite-filled microvein within the fracture corridor. The 'joints' within the fracture corridor tend to die out into (probably chlorite) microveins <1 mm thick, and to have chlorite coats on exposed fracture surfaces. This suggests that the joints in the fracture corridor consists of weathered-out microveins.

main gneissic foliation is also defined by chlorite, and by grain shape preferred orientation in potassium feldspar, albite and quartz. Ilmenite is also aligned within the gneissic foliation. The microveins show trace amounts of calcite and titanite developed at what appear to be extensional bends with a slight component of shear. Chlorite suggests the microveins formed under amphibolite or greenschist facies conditions (e.g., Fettes et al. 1985; Glodny, Kühn, and Austrheim 2008).

3.2 | Sundtsveg 47

This ~0.8-m wide apparent joint swarm is in gneiss with a foliation that dips ~17° towards 112°, and consists of at least 12 joints that dip ~77° towards 001°. The joints show a dark green coating and are seen to pass into <2 mm wide microveins of dark green mineral.

3.3 | Telavåg

This location (Hermansen 2019) shows a gneissic foliation that dips steeply to the north. Several apparent joint swarms are exposed, these having widths of up to ~20 m. The apparent joint swarms consist of steeply dipping joints that strike ~NNE-SSE. Some en-echelon joints occur and fracture frequency is up to 12 per metre (Hermansen 2019, figure 5.2–5). The joints are seen to pass laterally into microveins of a dark mineral, these being <2 mm thick.

Although it is likely that more of the apparent joint swarms in the area (Sanderson and Peacock 2019) are also formed from the weathering-out of microveins, it appears that exposures have to be fresh for the microveins to be observable. Both of the Sundtsveg locations are man-made exposures less than 5 years old (as of 2023), while the Telavåg exposure is on the coast facing the North Sea.

4 | Model for the Development of Apparent Joint Swarms

We suggest a model for the development of the apparent joint swarms described above involving the following sequence:

1. The host rock was weakened by a zone of unmineralised microcracks (Figure 4a), possibly caused by tectonic extension, thermal contraction or damage related to a nearby fault.
2. One of the microcracks propagated to form a longer and wider extension fracture (Figure 4b; Segall 1984). The role of microcracks and other types of flaw in the development of extension fractures is discussed by Fischer and Polansky (2006), with the mechanics of fracture development being discussed by, for example, Hoek and Martin (2014).
3. The extension fracture became filled by a mineral to form a microvein (Figure 4c) after it became connected to a fluid migration pathway. The migrating fluids precipitated material sourced from elsewhere and/or altered the host rock. While the microveins currently consist of chlorite, they may originally have been filled by a different mineral. This microvein was stronger (had a higher tensile strength) than the host rock, which was weakened by microcracks.
4. Another microcrack propagated to form an extension fracture (Figure 4d) and the cycle repeated. New extension fractures can follow earlier veins if the tensile strength of the vein is lower than the tensile strength of the host rock (e.g., Shang, Hencher, and West 2016). In this case, however, the tensile strength of the microveins was greater than that of the host rock. This is the *crack-jump process* of Caputo and Hancock (1999), who describe calcite microveins in Liassic limestones in Somerset, UK. Caputo and Hancock (1999) suggest that these are joints that were mineralised, with the microveins having greater tensile strength that the host rock, causing subsequent fractures to develop adjacent to the initial fracture. Also see Vass et al. (2014) for modelling of how vein fill may influence re-fracturing.
5. Eventually, a swarm of microveins developed (Figure 4e), which may be analogous to the 'tabular fracture clusters' of Riley and Tikoff (2010), which they attribute to dynamic

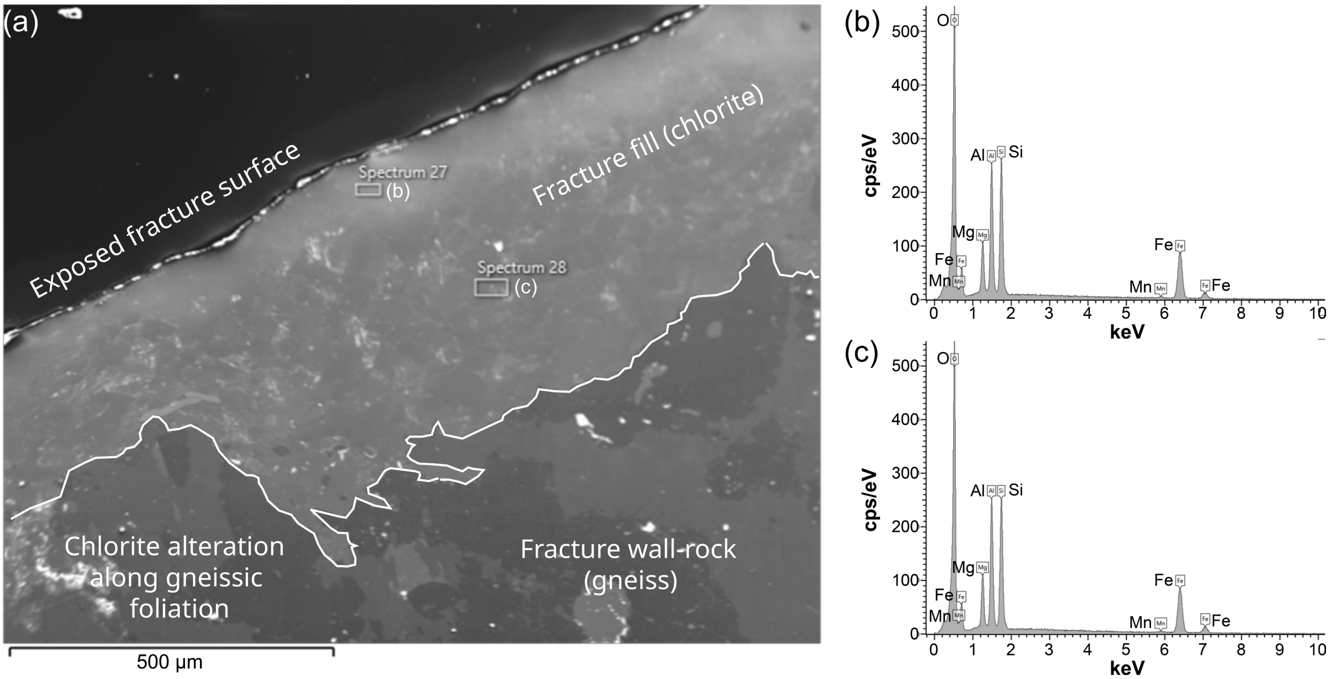

FIGURE 3 | (a) Scanning electron microscope image of the sample collected from Sundtsveg 172, Nesttun. The fracture appears to be filled by chlorite, with an irregular boundary, and some chlorite alteration along gneissic foliation. The locations of the two sampling points are illustrated. (b) Spectrum 27. cps/eV = counts per second/electronvolts, keV = kiloelectronvolts. (c) Spectrum 28, with the results being similar to those for spectrum 27.

fracturing in response to volatile overpressure from a granite porphyry.

6. The vein material was partly weathered-out (Figure 4f).

Note that this model is largely conceptual and requires significant additional constraints to test the validity. There is currently no meaningful information about the temperatures, stresses, fluid pressures and fluid chemistries during initial fracturing, during the development of microveins, or during the weathering-out of the microveins (see Bons et al. 2022, for a review of rock fracture mechanics). Furthermore, the original composition of the microveins, and therefore their mechanical properties, is currently unknown. Although the microveins are currently filled by chlorite, they may originally have been filled by another mineral (potentially with a higher tensile strength than chlorite or the host gneiss) and subsequently altered. For example, Glodny, Kühn, and Austrheim (2008) interpret chlorite in the Bergen area as having formed from amphibolitisation of omphacite, phengite and amphibole, while Centrella et al. (2018) describe replacement of clinopyroxene by chlorite during retrograde metamorphism. We do not, however, see evidence for retrograde reactions in the sample studied. It is also uncertain whether the mineral within the microveins was sourced from elsewhere or is the result of in situ alteration of the host rocks. Alteration of the gneissic foliation away from the fracture wall suggests that the chlorite in the micro-veins formed from local fluid-rock interaction along the fracture wall. They would therefore be *reaction veins* (e.g., Bucher 1998; Bégué et al. 2019).

There are also uncertainties about the fractures that form the apparent joint swarms. Several possibilities are suggested:

1. The model presented in Figure 4 is that the microveins have been partly weathered-out, and this interpretation appears to be supported by field observations of joints with chlorite-coated walls passing laterally or vertically into chlorite microveins. It is uncertain, however, at what depth that weathering occurred. This could be resolved if and when core data become available.
2. It is also possible that later joints follow the microveins, although this explanation does not explain why the joints would be so closely spaced. For example, Peacock, Sanderson, and Magán (2023) show examples of later joints following earlier calcite veins.
3. It is also possible that the apparent joints formed by subaerial or near-surface cracking along the microveins, either caused by erosion or by human activity. Two of the exposures described are man-made and the other faces the North Sea, so is subject to coastal erosion. We note that it is difficult to sample the microveins because the rock tends to break along the microveins, even when pulling out rock samples out by hand. It is therefore possible that some of the apparent joint swarms are ground-surface features.

5 | Discussion

We do not suggest that the model presented in Figure 4 explains *all* fracture corridors or joint swarms, not even all of those in the Bergen area. We have, however, identified several apparent joint swarms that appear to have developed from the partial weathering of microveins. We therefore suggest that, when analysing joint swarms elsewhere, care should be taken to check for evidence of microveins.

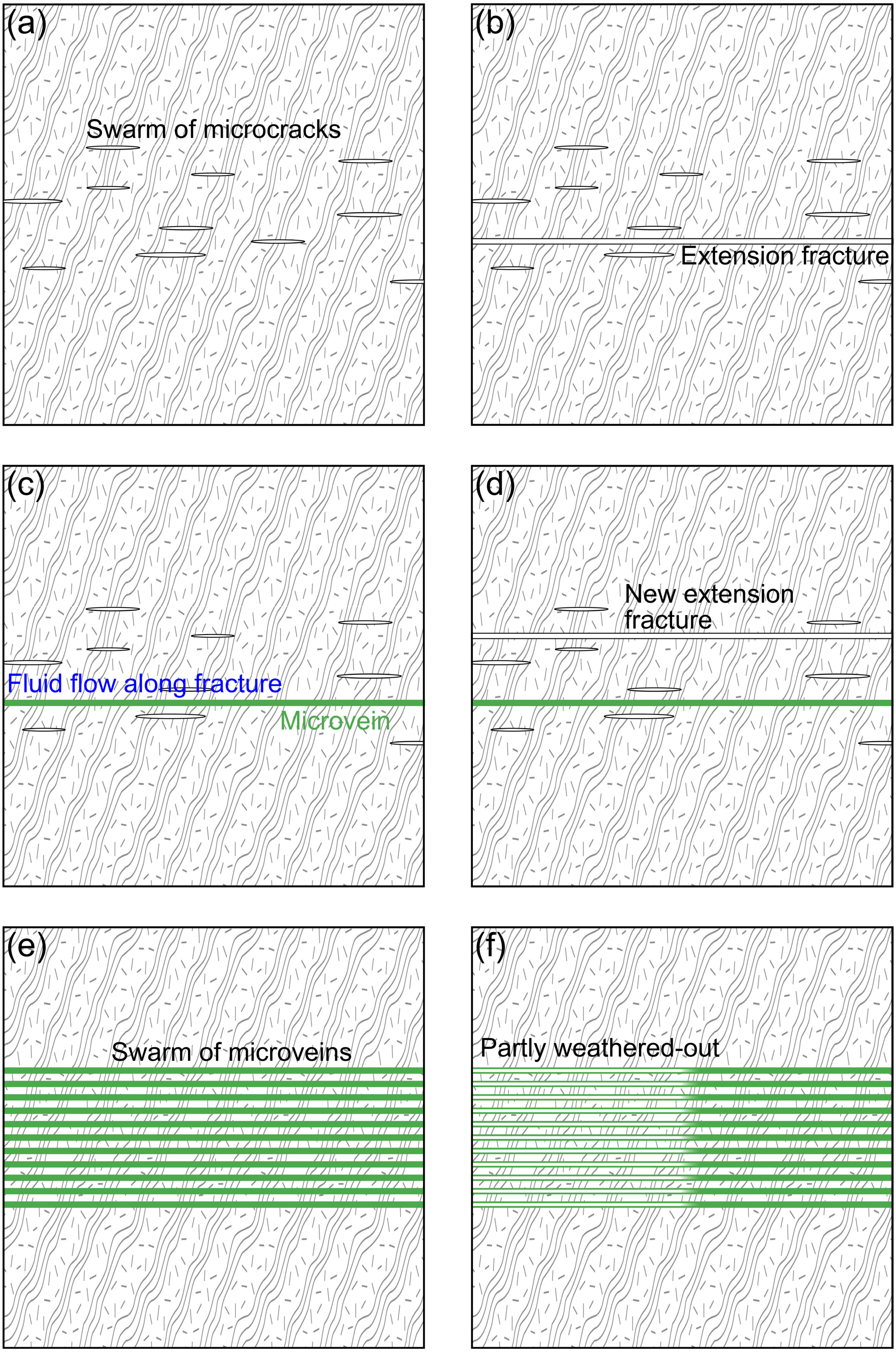

FIGURE 4 | Model for the development of apparent joint swarms. (a) A zone of microcracks developed in the gneiss. (b) A microcrack propagated as an extension fracture. (c) The fracture became connected to a fluid source that precipitated minerals in the crack to form a microvein. (d) That initial crack became stronger than another of the microcracks, which then propagated as an extension fracture (i.e., the crack-jump process). (e) The cycle was repeated until a swarm of microveins was developed. (f) Weathering (possibly near-surface) removed much of the mineral fill to produce what appears to be a joint swarm.

Gabrielsen and Braathen (2014) state that fracture corridors and joint swarms 'can be regarded to represent an incipient stage of faulting'. The calcite microveins in Liassic limestones in Somerset that are described by Caputo and Hancock (1999) are commonly clustered around faults, with Peacock (2004) suggesting they were precursor structures to faulting (although a fault may not have actually developed in some cases). It is possible that the fracture corridors around Bergen formed as precursory damage to faults, even though there do not appear to be any nearby faults and it is possible that faults may not actually have developed. It is also possible that other processes may be responsible for fracture corridors or joint swarms. It should not be assumed that a fracture corridor means that there must be a nearby unexposed fault.

While it is important to distinguish between veins and joints (e.g., Manda and Horsman 2015; Peacock and Sanderson 2018), there are situations in which the distinction may be ambiguous. For example, it is likely that some veins started as joints that were subsequently partly (e.g., Jacques et al. 2018) or completely mineralised (e.g., Caputo and Hancock 1999). Some authors (e.g., Rehrig and Heidrick 1972; Roberts 1979) use the term *mineralised joint* to imply a vein started as a joint, with Segall (1984, figure 2) showing a 'joint' filled with chlorite-epidote. Also, the fractures described here appear to be joints (i.e., they are relatively long, narrow unfilled extensional fractures) but are actually partly weathered-out microveins. The term *joint swarm* seems to be inappropriate if the fractures are not actually joints but partly weathered-out microveins, which is why we use the term *apparent joint swarms*.

Various papers describe the effects of fracture corridors on fluid flow in the sub-surface (e.g., Questiaux, Couples, and Ruby 2010; Furtado et al. 2022; Afroogh et al. 2023). If fracture corridors have been modified by such potentially near-surface processes as weathering or cracking of microveins, measuring apparent joint swarms in exposures may give a false impression about their characteristics in the subsurface. Apparent joint swarms at the surface may only exist as unweathered fracture corridors consisting of closed microveins at reservoir depths (e.g., Sanderson 2015; Martel 2017), which will not aid permeability in a reservoir. The existence of apparent joint swarms may indicate, however, that fracture corridors consisting of microveins may be effectively stimulated by chemical, thermal or hydraulic methods.

6 | Conclusions

A conceptual model is suggested for the development of apparent joint swarms in the gneisses around Bergen, which involves the following sequence: (1) initial extension took the form of a zone of microcracks; (2) one of the microcracks then propagated; (3) the propagated fracture linked to a fluid source that precipitated a mineral to form a microvein; (4) the microvein then had a higher tensile strength than the gneiss with microcracks, so another microcrack propagated as an extension fracture; (5) repetition of the cycle created a swarm of microveins; (6) much of the mineral fill was removed by weathering to produce an apparent joint swarm. It is possible, however, that some of the structures studied are the result of human-induced fracturing along microveins. This model does not explain all joint swarms, but it is suggested that joint swarms elsewhere should be analysed to determine whether they were influenced by microveins. It is suggested that care should be taken when using exposed analogues to predict reservoir behaviour because apparent joint swarms at the surface may not occur at reservoir depths.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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