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Review Article

Ageing of the Haematopoietic Stem Cell Compartment and Clonal Selection

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Abstract

Background: Ageing of the haematopoietic stem cell compartment is described as declining cell function, largely in mice, and as clonal selection measured in human blood.

Objective: To separate the evidence by species and by driver gene, and to ask what each body of work supports clinically.

Methods: Narrative review of population cohorts, clonal phylogenies, transplantation series and mouse experiments.

Findings: In 17,182 persons, clonal haematopoiesis rose from 5.6% at 60-69 years to 18.4% at 90 years and above. Growth rates differed by driver, from about 5% per year for DNMT3A to over 50% for SRSF2 P95H among 385 individuals. In 10 subjects, 30-60% of haematopoiesis over 75 years derived from 12-18 clones, a collapse that aged mice did not display.

Conclusions: The evidence supports driver-specific rather than pooled risk statements and labelling rejuvenation results as mouse work; it does not show that oligoclonal haematopoiesis is typical after 70.

Keywords: Haematopoietic stem cells; Clonal haematopoiesis; Ageing; Clonal selection; Somatic mutation

Introduction

Blood is renewed for a lifetime from a stem cell compartment that is itself changing. The changes are now described at two levels that were once treated as one: a decline in the functional properties of individual haematopoietic stem cells, and a reorganisation of the population structure of the compartment through somatic mutation and Darwinian selection. The first level is largely a mouse literature. The second is largely a human one, built on deep sequencing of blood DNA and, more recently, on whole-genome phylogenies reconstructed from single-cell-derived colonies. The two literatures are routinely written up as a single story, and the assumption that binds them — that mouse haematopoietic ageing is a compressed version of human haematopoietic ageing — has recently acquired a direct counter-example.

This review separates the evidence by species and by driver gene. Both separations matter clinically. Clonal haematopoiesis is not a single entity with a single risk figure; it is a collection of genotypes with growth rates that differ several-fold and disease associations that differ in kind. And a therapeutic proposal grounded in mouse transplantation biology is not the same object as an epidemiological association measured in tens of thousands of people [1,2].

Prevalence and the Age Gradient in Human Cohorts

The defining feature of clonal haematopoiesis is its age dependence. In whole-exome sequencing of 17,182 persons, detectable clonal haematopoiesis rose from 5.6% (95% CI 5.0-6.3) at 60-69 years to 9.5% (95% CI 8.4-10.8; 219 of 2,300) at 70-79 years, 11.7% (95% CI 8.6-15.7; 37 of 317) at 80-89 years, and 18.4% (95% CI 12.1-27.0) at 90 years and above; below 40 years the authors describe such mutations as very rare and report no numeric estimate [3]. Across the whole cohort, 805 candidate somatic variants in 73 genes were found in 746 persons, with DNMT3A accounting for 403 variants, TET2 for 72 and ASXL1 for 62

[3].

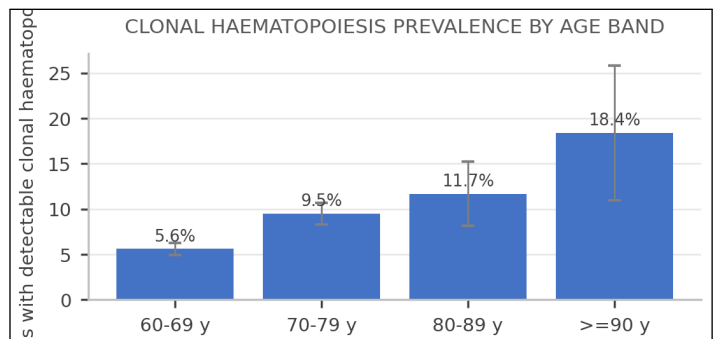


Figure 1: Prevalence of detectable clonal haematopoiesis by age band with 95% confidence intervals, from whole-exome sequencing at a variant allele fraction detection limit of 0.035; bands below 60 years are not plotted because no numeric estimate is reported for them, N = 17,182 [3].

Every one of these figures is conditioned on assay sensitivity. At a mean sequencing depth of 84 reads, the limit of detection for single-nucleotide variants was a variant allele fraction of 0.035 [3]. Prevalence estimates are therefore statements about clones large enough to be seen, and the conventional minimum variant allele fraction used to define clonal haematopoiesis of indeterminate potential is a reporting convention rather than a biological boundary [4,5].

A parallel Swedish study of 12,380 persons reproduced the gradient with independent calling criteria: clones with candidate drivers in 0.7% of those under 50 years and 5.7% of those over 65; clones with candidate or unknown drivers in 0.9% and 10.4% respectively; and clones defined only by carrying three or more mutations in 0.3% and 4.6% [6]. In tumour-free blood from 2,728 individuals in The Cancer Genome Atlas, more than 2% of individuals and 5-6% of people older than 70 years carried mutations representing premalignant expansion, with 77 blood-specific mutations in cancer-associated genes, 83% of them falling in 19 leukaemia and lymphoma genes and recurrence concentrated in DNMT3A, TET2, JAK2, ASXL1, TP53, GNAS, PPM1D, BCORL1 and SF3B1 [7]. Whole-genome sequencing of 11,262 Icelanders identified 1,403 cases and showed a germline contribution, the TERT deletion rs34002450 conferring an odds ratio of 1.37 (P =

7.4 x 10⁻¹²) [8]. The largest surveys give carrier counts without age stratification: 4,229 carriers among 97,691 TOPMed whole genomes [9] and 40,208 among 628,388 exome-sequenced individuals [10].

Driver Genes Are Not One Risk Category

Pooling these genotypes into a single exposure discards most of the information. Longitudinal resequencing of 1,593 blood samples from 385 individuals aged 55-93 years, at up to five timepoints spanning 3.2-16 years (median 12.9), resolved growth rates that differ by an order of magnitude: DNMT3A and TP53 clones expanded at roughly 5% per year, TET2, ASXL1, PPM1D and SF3B1 at roughly 10% per year, and SRSF2, PTPN11 and U2AF1 at 15-20% per year, with SRSF2 P95H exceeding 50% per year [11]. Clones carrying the same mutation varied around these central values by about plus or minus 5% per year, and only 58% of JAK2 V617F clones followed stable exponential growth at all, which is a caution against treating any single gene as a homogeneous exposure [11]. An independent longitudinal series — 248 samples from 85 individuals sampled roughly three-yearly from age 70 and age 79 over 12 years — likewise placed TP53, SF3B1 and SRSF2 above the commonly mutated JAK2 and DNMT3A on fitness, with U2AF1 Q157R at 33.5% [12]. Analysis of blood sequencing from approximately 50,000 individuals established that positive selection, not neutral drift, is the dominant force shaping these dynamics [13].

Growth rate is the defensible proxy for hazard, because per-gene hazard ratios for progression are not consistently established across cohorts. What is established is a qualitative gene ranking from pre-diagnostic sampling. Among 212 women healthy at baseline who developed acute myeloid leukaemia over a median 9.6 years of follow-up, mutations in IDH1, IDH2, TP53, DNMT3A, TET2 and the spliceosome genes raised the odds of leukaemia, and all 21 subjects with TP53 mutations and all 15 with IDH1 or IDH2 mutations progressed [14]. A second design compared 95 individuals sampled on

average 6.3 years before diagnosis with 414 matched controls, finding more mutations per sample and higher variant allele frequencies in the pre-leukaemic group, with the model validated in 29 further cases and 262 controls [15]. The practical reading is that the modal DNMT3A clone of an octogenarian and a slow-growing splicing-factor or TP53 clone in the same person are different clinical objects.

Table 1: Measured annual clonal growth rates by driver gene from longitudinal blood sampling, N = 385 individuals aged 55-93 years with 1,593 samples over a median 12.9 years, with one independent fitness estimate from a separate cohort of 85 individuals [11,12].		
Gene or variant	Growth per year	Source cohort
DNMT3A; TP53	about 5%	385 individuals
TET2; ASXL1; PPM1D; SF3B1	about 10%	385 individuals
SRSF2; U2AF1; PTPN11	15-20%	385 individuals
SRSF2 P95H	over 50%	385 individuals
JAK2 V617F	58% of clones stable	385 individuals
U2AF1 Q157R	fitness 33.5%	85 individuals

The Mouse Foundation and What It Supports

The mechanistic account of stem-cell ageing is a mouse account. Competitive transplantation established that ageing of the haematopoietic compartment is cell-intrinsic to the stem cell [16], and that lineage skewing arises through clonal expansion of pre-existing myeloid-biased stem cells rather than through the reprogramming of individual cells [17]. Single-cell RNA sequencing of aged mouse stem cells added a platelet and megakaryocyte bias to that picture [18]. Inflammatory signalling has been developed, again largely in animal and in vitro systems, as the mechanism that couples an ageing organism to selection within its stem cell pool [19]. The proposition that depleting myeloid-biased stem cells rejuvenates aged immunity is a mouse result with no human counterpart, and should not be presented as a therapeutic prospect without that qualification [20].

The methodological objection is that transplantation into an irradiated recipient is itself a perturbation, and that changes in downstream progenitors or in the marrow

microenvironment could produce the myeloid shift attributed to the stem cells [21]. The human counterpart to the mouse expansion result is a single cross-sectional immunophenotyping and xenograft study, which found that human marrow stem cells increase in frequency, lose quiescence and become myeloid-biased with age; the direction of that effect is what the study supports here, and its quantitative estimates could not be verified for this review and are therefore not reproduced [22]. No human competitive-transplantation experiment of the equivalent design exists or can exist.

The same asymmetry governs the cardiovascular story. Bone-marrow transplantation in mice showed that TET2-deficient clonal haematopoiesis accelerates atherosclerosis [23]. The human evidence is associational: across four case-control studies totalling 8,255 participants, carriers had 1.9 times the risk of coronary heart disease (95% CI 1.4-2.7) in the prospective cohorts and 4.0 times the risk of early-onset myocardial infarction (95% CI 2.4-6.7) in the retrospective cohorts, with DNMT3A, TET2, ASXL1 and JAK2 each individually associated [24]. Mouse mechanism plus human association is commonly read as human causation; that inference should be stated rather than assumed.

Clonal Diversity and Its Collapse

The most consequential recent human finding is structural rather than epidemiological. Whole-genome sequencing of 3,579 single-cell-derived colonies from 10 subjects aged 0-81 years showed that haematopoiesis in adults under 65 is massively polyclonal, the largest clades each contributing under 2% of blood production, whereas in individuals over 75 years, 30-60% of haematopoiesis derived from 12-18 independent clones contributing 1-34% each [25]. The same phylogenies place 20,000-200,000 stem and multipotent progenitor cells in even contribution in younger adults [25], consistent with the 50,000-200,000 estimated from 140 colonies of a single healthy 59-year-old man carrying some 129,582 called

somatic mutations [26]. Single-cell mitochondrial lineage tracing reproduced the collapse independently: expanded clades accounted for 3.4% and 8.7% of haematopoietic cells in two young donors versus 34.4% and 46.3% in two aged donors, with lower Shannon diversity in the aged; in one young donor, 78 distinct stem cell clonal groups were resolved and output differed 4.9-fold between the top and bottom clonal deciles [27].

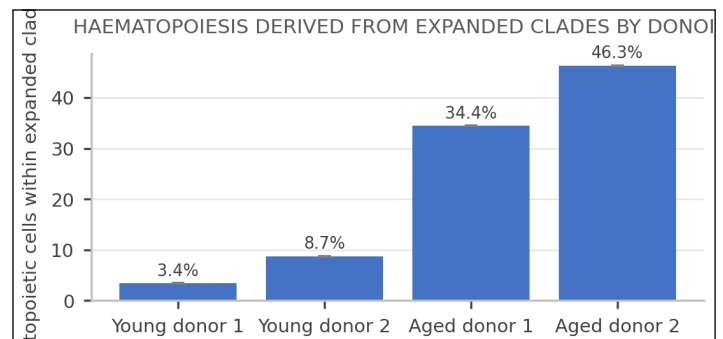


Figure 2: Proportion of haematopoietic cells falling within expanded clades in two young and two aged donors, by single-cell mitochondrial lineage tracing, N = 4 donors at roughly 11,000-15,000 cells each [27].

These effect sizes are large and mutually consistent, but they rest on 10 individuals [25] and four donors [27]. Between-individual variance is essentially unknown, and the correct statement is that diversity collapsed in the individuals studied, not that haematopoiesis becomes oligoclonal in most people after 70.

The species assumption fails precisely here. Whole-genome sequencing of 1,547 mouse colony genomes passing quality control — 908 stem cells and 639 multipotent progenitors from 1,845 colonies, at 3 and 30 months — showed a stem cell pool growing across life to a median of 72,414 cells (CI 25,510-98,540) and a somatic mutation rate of about 45 per year, roughly threefold the human rate of 14-17 per year. Yet aged mice did not display the profound loss of clonal diversity that characterises human haematopoietic ageing [28]. Whatever produces the human oligoclonal collapse is therefore not a slower version of what mice do, and mouse ageing phenotypes cannot be recited as human mechanism.

Transplantation as an Accelerated-Ageing Experiment

Allogeneic transplantation transfers a stem cell compartment of known age into a new host and is the closest available human analogue of a competitive repopulation assay. In registry data from 6,349 training and 4,690 validation donor-recipient pairs, each 10-year increment in unrelated donor age raised the hazard ratio for overall mortality by 5.5%, with best survival from HLA-matched donors aged 18-32 years ($P < .001$) [29]. Donor clonal haematopoiesis is common: 92 mutations with median variant allele frequency 5.9% in 80 of 500 related donors aged 55 or older, whose recipients had more chronic graft-versus-host disease but less relapse and similar survival [30]; and 22.5% of 1,727 donors aged 40 or older, with DNMT3A at 14.6% and TET2 at 5.2%, where higher-fraction DNMT3A clones were associated with improved recipient survival through reduced relapse while donor cell leukaemia arose instead from TP53, splicing-factor or germline DDX41 lesions [31]. This is the driver-gene distinction made clinical: the common clone is not the dangerous one.

Long-term follow-up shows the cost to the graft. Among 42 related donor-recipient pairs at a median of 16 years, 23 of 84 individuals (27.4%) carried clonal mutations, 23.8% of donors and 31% of recipients, with five cases of donor-engrafted clonal haematopoiesis and one progressing to myelodysplastic syndrome in both members of a pair; recipient telomeres had shortened by the equivalent of about 20 years of additional proliferative history [32]. Phylogenies from 2,824 colony genomes across 10 sibling pairs sampled 9-31 years after transplant estimated 5,000-30,000 engrafted and still-contributing stem cells from donors aged 18-47 at transplant, roughly tenfold fewer from donors aged 50-66, and recipient clonal diversity reduced by the equivalent of 10-15 years of extra ageing [33]. In the autologous setting, 120 of 401 patients with non-Hodgkin lymphoma carried a qualifying somatic mutation at transplant; their 10-year overall

survival was 30.4% against 60.9% (HR 1.8, 95% CI 1.4-2.8) and their 10-year incidence of therapy-related myeloid neoplasm 14.1% against 4.3% ($P = .002$) [34].

Table 2: Clonal haematopoiesis and donor age in the transplant setting across registry, related-donor, long-term follow-up and autologous series [29,30,31,32,33,34].			
Setting	N	Prevalence	Key outcome
Unrelated donor registry	6,349 + 4,690 pairs	not assessed	5.5% higher mortality HR per 10 y donor age
Related donors >=55 y	500 donors	80/500 (16.0%)	More chronic GvHD, less relapse, similar survival
Donors >=40 y	1,727 donors	22.5%	High-VAF DNMT3A improved survival via less relapse
Pairs at median 16 y	84 individuals	23/84 (27.4%)	5 donor-engrafted clones; 1 progressed to MDS
Sibling grafts 9-31 y on	10 pairs	not the endpoint	Recipient diversity aged by 10-15 extra years
Autologous, lymphoma	401 patients	120/401 (29.9%)	10-y survival 30.4% vs 60.9%, HR 1.8 (1.4-2.8)

Clinical Consequences and Their Magnitudes

Progression to overt malignancy is uncommon but strongly patterned. Carriers had a hazard ratio of 11.1 (95% CI 3.9-32.6) for incident haematologic cancer, with absolute conversion at approximately 0.5% per year overall and approximately 1% per year among those with variant allele fraction above 0.10 [3]; the Swedish cohort gave a hazard ratio of 12.9 (95% CI 5.8-28.7) and 1.0% per year [6]. Non-malignant outcomes carry smaller relative risks over a far larger exposed population: all-cause mortality hazard ratio 1.4 (95% CI 1.1-1.8), coronary heart disease 2.0 (95% CI 1.2-3.5), ischaemic stroke 2.6 (95% CI 1.3-4.8) and type 2 diabetes odds ratio 1.3 ($P < 0.001$) in one cohort [3], and all-cause mortality 1.4 (95% CI 1.0-1.8) in the other [6]. A large multiple of a small hazard is where the population burden of clonal haematopoiesis mostly sits [5].

Conclusion

Three conclusions follow from separating the evidence rather than pooling it. Human haematopoietic ageing is

characterised by a loss of clonal diversity in the eighth decade that has been measured directly, at high resolution, in very few people; the phenomenon is robust within those individuals and its population distribution is unmeasured [25,27]. The mouse literature that supplies the mechanism does not reproduce that collapse, so mouse results must be labelled as such and used for mechanism rather than for extrapolated human trajectory [28,21]. And the clinical meaning of a clone depends on its driver: growth rates spanning 5% to over 50% per year, and gene-specific progression patterns in pre-diagnostic cohorts, make a single prevalence-based risk statement unusable at the bedside [11,12,14]. Transplantation supplies the one human system in which stem-cell age, clonal composition and outcome are measured together, and it consistently shows that donor age and donor clonal architecture are transmissible properties of a graft [29,33]. Interventions aimed at the clonal composition of the ageing compartment remain animal work [20], and the framework for interpreting them is now a decade old and still consolidating [2,1,19].

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