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Review Article

Immune Memory Based Classification of Human Herd (IMCHH)

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Abstract

Individual and population/herd immunity are at most depend on the nature of effector cells on first encounter and immune memory cells on re-infection or boost vaccination. The present opinion paper tempts to suggest human herd classification on the bases of immune memory cell nature. The immune memory stratification starts as; baseline immune memory function, innate immune memory, infection induced memory, pre-existing and current immune memory and vaccine induced memory. Based on this stratification and in accordance with; five classes can be proposed as; herd homeostatic immunity, herd trained immunity, herd natural immunity, herd mixed or hybrid immunity and herd vaccine immunity. For herd homeostatic immunity HHI, low grade innate and adaptive memory cell driven by environmental or endogenous microbiomes. Herd trained immunity HTI driven by innate immune memory cells and confinement, natural herd immunity (Infection), herd mixed or hybrid immunity HMI initiated by past infection or past vaccination and current immune memory of innate and adaptive nature as well as vaccine herd immunity VHI mediated by effector and memory B or T lymphocytes primed by vaccine antigens. The paradigm of classification of herd immunity starts as; dipartite, tripartite and reach this point of penta-partite classification systems. The present suggestion is being novel in its context

Keywords: Confinement; Herd; Hybrid; Homeostatic; Immunity; Memory; Lymphocyte

Introduction

The cornerstone of re-infection and recall or booster vaccine immunity both in individuals and herd immunity are the immune memory cells. These subsets of the immune cells encompasses; innate trained macrophages, trained NK cells, adaptive memory B-cell and adaptive memory T-cells [1,2]. WHO classification to herd immunity has been of two classes; natural herd immunity and vaccine herd immunity [3,4]. Recently, a class of herd immunity has been added to the herd trained immunity [5-7]. The status of the present classification is rather inconsistent. The objective of the present opinion paper was to suggest a new classification system of herd immunity.

Immune Memory: Basic Core Concept

The immunologic memory IM of human and animal beings are based mostly on subsets of; macrophages, B-cells and T-cells. IM is essentially of two types non-specific conferred by macrophages and specific conferred by either of B or T-cells or both. In other word it can be of innate trained immune memory and adaptive immune memory.

On training macrophage it shift from its normal immune state to more mature, pro-inflammatory intermediate CD14++ CD16++ or to CD14+CD16++ phenotype. It expresses enhanced capacity to interact and present antigens to T-cells (HLA-DR) upregulated.

As well as increase of integrin expression or facilitating better adhesion, migration into tissue and phagocytosis. They provide strong co-stimulatory signals to adaptive immune cells during contact with (CD80/CD86). Their TLR 4 and dectin-1 are upregulated leading to hyper-sensitizing of the cells to detect forthcoming LPS. In training NK cells through CMV infection or combination of Il12/IL15/IL1B undergo a distinct surface marker remodeling. In which Ly49 receptor and NKG2D are activated to recognize viral infections [16-18]. Innate immune memory IIM are of two types. Central and trained in bone marrow though immune-metabolic and epigenetic reprograming and peripheral is trained in peripheral tissues. It operates in vaccine non-specific protection such as that of BCG in COVID-19 [1,19].

Memory B-cells are of two subsets, classical memory B-cell with high affinity BCR due to somatic hyper-mutation rapidly differentiated into plasma cells. Generated in lymph node germinal centers during T-cell dependent immune responses. Requires CD40-CD40L interactions. While, the tissue resident memory B-cells are localized in peripheral tissues provide rapid local antibody responses. Sustained antigen exposure or repeated stimulations in tissue niches enriched with survival factors, Table-1.

Subsets	CD19	CD24	CD27	CD38	CD40/CD40L	IgG	IGD
Naïve B cells	+	+	-	+	-	-	-
Effector B cells	+	-	-	+	-	-	+
Classical memory B cells	+	-	+	+	+	+	-
Tissue resident memory B cells	+	-	+	-	-	-	+

Table 1: Memory B-cells surface markers {antibodies, 2025,20].

Memory T-cells, these memory cells are of three subsets. As central memory T-cells TCMs, effector memory T-cells and resident memory T-cells. TRMs express CD62L anCCR7 (lymphoid homing), high proliferation capacity and delayed effector functions. Derived from naïve or effector T-cells, requires IL7, IL15 for survival and quiescence. TEMs lack CD62L and CCR7 (migrate to tissues), rapid cytokine production upon reactivation, Table 2. Derived from effector T-cells, lose lymphoid homing markers but retain effector plasticity. TRMs, persist in non-lymphoid tissues, express CD69 and CD103 for tissue retention. Imprinted by tissue specific signals (e.g. TGFB), formed during immune response resolution [21].

Cell Type	CD44	CD62L	CCR7
Naïve T-cells	Low	++	+
Effector T-cells	+	Low	-
Effector memory T-cells	+	Variable	-
Central memory cells	+	+	+

Table 2: Memory T-cell markers [24].

Immune Memory Cell Stratification

Immune memory cells are being suggested as a base for classification of herd immunity. Memory stratification starts with the basal homeostatic minimal memory cell counts induced by environmental or endogenous microbiome mainly of an adaptive nature, like memory B and memory T-cells in which both memory and effector immune cells forms the baseline immune function of an individual and population. In this suggestion it forms the first immune memory stratum and the base of class I herd immunity [4].

Innate immune memory, the central type forms the base of second stratum of memory cell in which trained macrophages and/or natural killer cells undergoes central training of these cells in bone marrow through epigenetic and metabolic reprograming, then released to circulation. This stratum found in special circumstances of human social confinement, microbiome dysbiosis and restriction as well as immune suppression due to viral pandemic. On relax and after a while from confinement herd infection upsurge with S. pyogenes infection in an epidemic episode [5-7].

The possible fraction or fractions of the herd undergoing natural infection may induce memory and effector cells. Such memory cells as B or T-cells may form the base of the third memory stratum level. Highly virulent epidemic infections does not leave windows for memory cell formation [3].

Past infection or past vaccination may leave waned mediator and/or a level of immune memory cells constituting the state of pre-immunity of preexisting immunity. On infection or vaccination the pre-existing memory cells may forms an add on effect with current infection or vaccination such mixed memory cell activation may form the base for the fourth memory cell stratum level. Or it can be of downregulating effect which holds the position out of this concern [23-25].

Vaccine induced and adaptive memory B or memory T-cells in a human population constitute the basics for the stratum five level of memory cell stratification system. This memory level effectivity depends on vaccine coverage level attack rate among vaccinee and non-vaccinee as well as herd threshold [3,26-29].

Memory cell stratification as related to herd immunity classification system. Stratum I have not ever been considered as a class in an old and current publications as evident in dipartite and tripartite classification of human herd immunity. Innate trained immune memory has been introduced recently by a Polish scientists as a third class and not known in the dipartite system [5-7]. Strata III and V of the immune memory were forming the basis of dipartite classification, although they don't even mention it as having such role. The penta-partite system, the subject of present theme covers most of the expected cases that face the human herd and is being novel suggestion for the first time in classification of human herd immunity, Table-3.

Memory Strata Nature	Level	Penta-Partite Classification	Tripartite Classification	Dipartite Classification
Homeostatic herd immune memory	I	I	-	-
Trained herd immune memory	II	II	III	-
Natural infection herd immune memory	III	III	I	I
Hybrid or mixed immune memory	IV	IV	-	-
Vaccine herd immune memory	V	V	II	II

Table 3: Immune memory based stratification in human herd and its relation to human herd classification.

Herd

Herd is a group of human or animal beings that lives in same ecologic niche, where, they affect the niche and the niche affect them in mutual affection pattern. The immunological functions of the group are encoded by the major histo-compatibility complex of the short arm of chromosome 6 in man. Variation in genes, proteins and/or cells within an individual take part in the overall variations in human herd. Herd variation are facing concurrent; past, present and forthcoming infections and/or vaccination. Vaccination and infections are constituting the basic stimulants for the immune conversion from naïve B or naïve T-cells or both into effector and memory cells [9].

Herd Immunity (HI)

HI is the overall immune threshold established after vaccination of part of the herd in view of protecting the other part. Herd immunity developed in either of two ways; infection and vaccination. Infection induced herd immunity is currently not advisable

by WHO due to the predicted infection burden in the herd that may occur. Mass vaccination is advisable by WHO. WHO classified herd immunity into natural and vaccine induced herd immunity [3]. Innate immune cell can be of importance in herd immunity in special cases of pandemic post confinement infection upsurge [5-7]. In this opinion paper a suggestion of new classification system is being presented. Vaccination induced herd immune response gave sectors of non-response, mild response, moderate response and high response. Herd immunity threshold and vaccine efficacy can be calculated in the following mathematical formulas [10,16].

$$\text{Vaccine Efficacy} = \frac{\text{Infection rate in non-vaccinated} - \text{Infection rate in vaccinated}}{\text{Infection rate in non-vaccinated}} \times 100$$

Herd Immunity Threshold = 1 – 1/Ro, where Ro is reproduction number.

Homeostatic Herd Immunity

Normal healthy human individuals forming the healthy herd exhibits immune homeostasis, symbiotic microbiome and baseline humoral and cellular immune functions. These encompassing normal levels of cytokines, antibodies and normal immune cell counts including memory B, T-cells driven through the induction of environmental and endogenous symbiotic microbiomes. Memory immune cell are constituting the pillars of the homeostatic herd immunity [4]. The basic characteristics of this class was depicted in the followings.

Stimulant: Environmental and endogenous microbiome activities

Immune Response Nature: Humoral and/or cellular

Immune Memory Cells: Minimal levels of adaptive memory T and/or memory B cells.

Measurement Approaches: Monoclonal antibody specific for surface markers of both T (CD44, CD61L CCR7) and B-cells (CD19, CD27, CD38, CD40/CD40L, IgG, IgD). Or flow cytometry studies.

Herd Threshold: minimal non-specific.

Novelity: Use of homeostatic immune memory cells in classification of herd immunity.

Herd Trained Immunity

Herd trained immunity HTI is the reduction or deprivation of herd immunity under the conditions of confinement and social restriction for long time in human being, normal gut microbiome undergoes changes in composition, diversity, richness and function, a state of dysbiosis. In which cytokine signaling with intestinal immune cells, qourum sensing signaling in between microbiome components are deprived leading to a state of immunosuppression in the gut micro-environment which may influence the microbiome-bone marrow-axis. Such herd immune events may render individuals within this confined herd susceptible to microbial population infections in an epidemic episodes. This state is termed as “Immune Debt” [12,13]. When the confinement period relaxed and opened to normal usual daily life, the microbiome gradually gained its balance state and the immune homeostasis restored, the state of immune debt gradually converted to normal homeostatic state. The herd immunity under the confinement and on normal daily life gained is termed as Herd Trained Immunity.

During covid-19 there has been a confinement, restriction of individuals mobility, restriction of gut microbiome-environmental microbiome interaction and immunosuppression state facing human populations across the globe. Such collective factors influence gut microbiome dysbiosis encompassing change in signaling with intestinal immune cells, change in quorum sensing signaling among entities of microbiome, limitation of diversity and herd immune deprivation. Taken together, these changes lead to evolution of pathobiont that can be of passing across damaged intestinal barriers to blood or lymph circulations followed by reaching bone marrow facing there the bone marrow leukocyte progenitors of the innate immunity leading to immune-metabolic and epigenetic reprograming of these innate cells to be trained forming innate trained immunity. Then the trained

macrophages trafficked to oral cavity through blood circulation of the individuals forming the herd and developing herd trained immunity HTI.

Herd trained immunity HTI become reduced during covid-19 lock-down renders the population susceptible to group A streptococcal infection in a massive episode patterns all over the world immediately after relaxing the confinement state of human herds, latter on the levels and episode form of GAS, Table 4, gradually vanished across the globe returning to the normal level matching that of before confinement [5,6,7,13-16].

Sequence	Infection Events
1	Pandemic confinement
2	Impaired immunity of individuals to long covid-19
3	Reduction in the limits of exposure to environmental microbiomes
4	Reduction in personal mobility
5	Reduction in immune silencing ability of the herd
6	Gut dysbiotic microbiome
7	Impaired cytokine signaling of gut microbiome and intestinal immune cells
8	Microbiome forming components change their quorum sensing signaling among each other
9	Deprived her trained immunity
10	Emergence and re-emergence of bacterial virulence factors and appearance of high virulent strains
11	Increase in respiratory viral infections which may predispose to secondary bacterial infection ,infection upsurge

Table 4: Sequences of infection upsurge events post to covid-19 pandemic confinement [5-7].

The characteristic features of this class are as in the followings:
Stimulant: Pandemic confinement, change in microbiome and limitation of co-reaction with environmental microbiome, immune suppression, upsurge if bacterial virulent pyogenic infections

Nature of Immune Response: Humoral mainly and/or cellular.

Measurement Approaches: Detection of trained memory macrophage cell surface markers (CD14, CD16) through monoclonal antibody and immunofluorescence or via flow cytometry.

Herd Threshold: Non-fixed depends on intensity and continuity of microbial stimulation across the population.

Novelity: The use of trained innate immune cell in classification of human herd immunity.

Natural Herd Immunity
When, normal virgin human herd contracts an infection the resulting immunity known as natural herd immunity. If significant portion of the population survive the infection outcomes, the developing specific antibody response and the immune memory arise. This portion may renders the other portions immune to that infection. Though, such herd immunity is not advisable by WHO due to the threat of possible highly virulent infection may cause massive death in the herd [3]. The basic characteristic features of this class is as in the followings:
Stimulant: Natural human herd epidemic infection.

Immune Response Nature: Humoral and/or cellular.

Memory cell type: In case of surviving herd infection the cell type can be memory B or memory T-cells.

Measurement Approaches: Detection of memory B-cell surface markers (CD19, CD27, CD38, CD40/CD40L, IgG, IgD) or T-cells (Cd44, CD62L, CCR7) via immunofluorescence with monoclonal antibodies or flow cytometry.

Heed Threshold: Infection survivor may not reach the limits of immune herd.

Novelity: The use of infection induced adaptive memory cells in classification of human herd immunity.

Mixed Or Hybrid Herd Immunity

The immune response in normal healthy individual forming the herd to natural microbiome components showed net result of either immune tolerance or baseline memory immune cell counts and baseline immune mediators (antibodies, cytokines). Such outcomes are finalized by baseline immune function. Post-infection, post-vaccination antibodies or cytokine were gradually waning, while memory cells were housed in an immune tissue micro-environment and to lesser extent migratory in blood stream or mucosa associated lymphoid tissues. On recall, a new homologous or heterologous antigen, such homed and/or migratory memory cells initiated and accelerated to produce immune response mediators. The level of such produced mediators can be with an add on effect "hybrid immunity". In other word upregulation for both of previous and new memory cell produced. Or downregulation that leads to deprivation of the immune response. Some herd past Salmonanella infection or past vaccination upregulate the immune response outcomes. Other viral infections may downregulate the immune response. This immune phenomenon is known as pre-immunity or pre-existing hybrid immunity [11,23-25]. The main characteristic features of this class are as in the followings:

Stimulants: Past-infection, past vaccination, mixed with current infection or current vaccination.

Immune Response Nature: Humoral mainly with up and/or down regulation as higtheining or suppression. It can be cellular.

Memory Cell Nature: Mixed type adaptive immune memory cells of either B or T-cells.

Measurement Approaches: Detection of memory specific cell B-cell markers (CD19,CD27,CD38,CD40/CDL,IgG,IgD), Memory T-cells (CD44,CD62L CCR7) by either monoclonal antibodies specific immunofluorescence or through flow cytometry.

Herd Threshold: Depends on vaccine coverage and limits of pre-existing immunity and can be sestimated by the formula

HITh = 1- 1/Ro where Ro equals production number.

Novelity: The use of pre-existing and current memory immune cells in classification of herd immunity.

Vaccine Herd Immunity (VHI)

Vaccine Herd Immunity (VHI) is the fraction or fractions of vaccinated individuals among the herd whom are vaccine immune against certain infection that make other non-immune individuals among the herd to be converted to immune. The immune response patterns of the individuals forming the herd can be sectored to; non-immune, low, moderate and high immune response. Vaccine herd immunity plots are either of simple positive linear or simple negative linear plots as regression analysis is indicated. The vaccine herd immunity is encoded by the gene complex, major histocompatibility complex MHC found on the short arm of the chromosome 6 in human beings [3,4,26-29]. The basic characteristic features of this class are as in the followings:

Stimulants: Vaccines

Immune Response Nature: Humoral and/or Cellular.

Memory Cell types: Memory T, memory B and/or trained memory macrophage.

Measurement Approaches: Detection of memory cell surface markers of B (CD19<CD27, CD38, CD40/CD40L, IgG, IGD) or T-cells (CD44, CD62L, CCR7) or macrophages (CD14, CD16) via specific monoclonal antibodies and immunofluorescence or flow cytometry.

Herd Threshold: Requires critical percentage of vaccine coverage among the other to provide herd immunity and can be calculated through the following formula;

HITh = 1-1/Ro where Ro equals production number.

Novelity: The use of adaptive immune memory cells in classification of human herd immunity.

XI- CLASSIFICATION OF HERD IMMUNITY:

From the aforementioned paragraphs immune memory oriented herd immunity, now human herd immunity ensemble in five classes as; i-homeostatic herd immunity, ii-herd trained immunity, iii-natural herd immunity, iv-mixed or hybrid herd immunity and v-vaccine herd immunity. These classes were briefly described as following Table-5.

I- WHO classification 2020 [3,4] i-Natural Her Immunity ii-Vaccine Herd Immunity
II- Update Classification,2026 [3-7] i-Trained Herd Immunity ii-Natural Herd Immunity
III-Current Proposed Classification System: Immune Memory Oriented,2026. i-homeostatic herd Immunity ii-Herd trained Immunity iii-Natural Herd Immunity iv- Mixed or Hybrid Herd Immunity v-Vaccine Herd Immunity

Table 5: Herd immunity classification systems.

Discussion

Recently, I came across three serial mono-authored publications that starts in 2024 lasted till 2026 [6,7,8]. They were the initial spark that passed into the built in circuit of the personal passion engine for this idea. The basic philosophy of it depends on using immune memory cell nature as a fundamental for classification of human herd immunity. WHO [3] made the classification into natural and vaccine herd immunity. The worker add a third class of human herd immunity based upon innate trained immunity [5-7]. The present classification system was not ever been reported. It starts with homeostatic herd immunity class I, herd trained immunity Class II, natural or infection herd immunity III, mixed or hybrid herd immunity class IV and vaccine herd immunity Class V. The basic immune-biologic characteristics of these classes were presented showed the following features:

i- immune memory cell type involved in.

ii- The immune memory stratification of these classes starts as; baseline natural immune memory function, innate immune memory, infection induced memory, pre-existing and current memory cells and vaccine induced current memory cells.

iii- limits of past exposure to either infection or vaccination.

iv- The eco-immunologic niche type that harbor the herd whether confine or non-confine.

v- herd history of virginity limits; virgin, non-virgin, precision of herd record.

vi- The practical suggested protocols for scoring classes were; herd history, herd threshold, herd vaccine efficacy, types of cell mediators involved, type of memory cell subsets involved and the immune detection protocols for scoring each of which.

Conclusion

Innate trained immune memory and adaptive immune memory cells served as valid bases for classification of herd immunity. The theme starts as dipartite (WHO) classes, tripartite classes and present proposal of penta-partite classification system.

Knowing that they do not mentioned the role of memory cell stratification as a bases in the classes they made. It seems that it constitutes a novel classification system in its context as compared to dipartite and tripartite systems.

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The author declare that no artificial intelligence AI were used in the preparation of this manuscript.

Authors' Contributions
All authors contributed equally to this paper.

References
1. Shnawa IMS. Potentials of innate immune memory. Br J Healthc Med Res. 2026;13(2):24-35.
2. Shnawa IMS. Microbial immunology. India-UK: BPI. 2025.
3. World Health Organization. Coronavirus disease: herd immunity, lockdowns and COVID-19. Geneva: World Health Organization. 2020.
4. Jenkins E, Latta C, Le HA. Immune cell interactions in development, homeostasis and immunity. J Cell Sci. 2025;138(18):jcs264374.
5. Marcinkiewicz J. Increase in the incidence of invasive bacterial infections following COVID-19 pandemic; potential link with decreased herd trained immunity-a novel concept in medicine. Pol Arch Intern Med. 2024;134(9):1-7.
6. Marcinkiewicz J. Post-pandemic upsurge of group A streptococcus infections: potential link to impaired herd trained immunity following COVID-19 lockdowns. Front Immunol. 2025;16:1684332.
7. Marcinkiewicz J. Herd trained immunity: a hidden component of population-level innate immunity. Arch Immunol Ther Exp (Warsz). 2026;74:17:1-7.
8. Shnawa IMS. Immune tuberculous anergy. J Pharm Res Int. 2026;38(3):6-12.
9. Shnawa IMS. Individual variation and human herd immunity. Nat Sci Res. 2014;4(8):31-8.
10. Randolph HE, Barreiro LB. Herd immunity: understanding COVID-19. Immunity. 2020;52:737-41.
11. Roberts GC, Glaser L. Hybrid immunity: a combination of vaccination and prior infection probably offer the best protection against COVID-19. Vaccine Work. 2022.
12. Needle RF, Russell RS. Immunity debt, a gap in learning or immune dysfunction? Viral Immunol. 2023;36:1-2.
13. Munro AP, House T. Cycles of susceptibility: immune debt explains altered infectious disease dynamics post-pandemic. Clin Infect Dis. 2024;81:1173-6.
14. Netea MG, Quintin J, van der Meer JWM. Trained immunity: a memory for innate host defense. Cell Host Microbe.

2011;9:355-61.
15. Netea MG, Ziogas A, Benn CS, Curtis N, van Crevel R, Dowling DJ, et al. The role of trained immunity in COVID-19: lessons for the next pandemic. Cell Host Microbe. 2023;31(6):890-901.
16. Chen J, Gao L, Wu X, Fan Y, Liu M, Peng L, et al. BCG-induced trained immunity: history, mechanisms and potential applications. J Transl Med. 2023;21:106.
17. Ifrim DC, Quintin J, Joosten LAB, Jacobs L, Jansen T, Jacobs C, et al. Trained immunity or tolerance: opposing functional programs induced in human monocytes after engagement of various pattern-recognition receptors. Clin Vaccine Immunol. 2014.
18. Kovalenko EI, Streltsova MA, Kanevskiy LM, Erokhina SA, Telford WG. Identification of human memory-like NK cells. Curr Protoc Cytom. 2017;79:9.50.1-11.
19. Shnawa IMS. Vaccine race, vaccine immunity and vaccine herd immunity. Clin Med Invest. 2022;5:1-4.
20. Syeda. B-cell memory: from generation to reactivation; a multipronged defense wall against pathogens. Cell Death Discov. 2024;10:117.
21. Giri S, Batra L. Memory cells in infection and autoimmunity: mechanisms, function and therapeutic applications. Vaccines (Basel). 2025;13(2):205.
22. Shnawa IMS. Vaccine technology at a glance. United Kingdom: Boffin Assess. 2019.
23. Li ZN, Liu F, Jung YJ, Jefferson S, Holiday C, Gross FL, et al. Pre-existing cross-reactive immunity to highly pathogenic avian influenza 2.3.4.4b A(H5N1) virus in the United States. Nat Commun. 2025;16:10954.
24. Ye C, Zhang X, Qiu L, Ji J, Jueqing G, Jia H, et al. Pre-existing Th1 immunity outperforms age in predicting antibody responses to SARS-CoV-2 inactivated vaccines. Adv Sci (Weinh). 2026;13(6):e14147.
25. Saxena M, Van TTH, Baird FJ, Coloe PJ, Smooker PM. Pre-existing immunity against vaccine vectors-friend or foe? Microbiology (Reading). 2013;159(1):1-11.
26. Medley GF. Herd immunity confusion. Lancet. 2020;396:1634-5.
27. Launer J. Revisiting herd immunity. BMJ. 2025;388:r199.
28. Schaeffer B, Taylor B, Bushman M, Hanage WP. The devil in the details: herd immunity and pandemic response. Cell Host Microbe. 2021;29(7):1048-51.
29. Tankeshwar A. Herd immunity: types, threshold, usefulness. Microbe Online. 2025.

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